The
AMERICAN JOURNAL
of
MEDICAL TECHNOLOGY

VOLUME 15 SEPTEMBER, 1949 NUMBER 5

THE EFFECT OF THE AGE OF SERUM SAMPLES AND OF ALIMENTARY LIPEMIA ON THE THYMOL TURBIDITY TEST*

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The liver has many functions, and many tests have been devised to measure these specific capacities. To date there is no single test that will measure hepatic function in all its aspects. To obtain a clear insight of the disturbances in the efficiency of the liver, several tests must be employed. The thymol turbidity is one of the newer liver function tests and is done in our laboratory as a routine procedure.

The test outlined by Maclagan[1] describes the use of a barbital buffer of pH 7.8 saturated with thymol. One part of blood serum is added to sixty parts of thymol buffer and the turbidity measured by the Kingsbury turbidity standards. To make the procedure practical for use with photoelectric methods several modifications have been developed. This laboratory is using a curve set up with barium sulfate standards,[2] and the results are recorded in units equivalent to the Kingsbury turbidity standards. Several workers have reported the use of thymol buffer with lower hydrogen ion concentration than pH 7.8. The tests herein described were made with thymol buffer of pH 7.68. All mixtures of thymol buffer and blood serum were allowed to stand at room temperature for thirty minutes before photoelectric evaluations were made.

The cause of the turbidity, or the precipitation, produced by

* Read before A.S.M.T. Convention, Roanoke, Va., June, 1949. Hillkowitz Memorial Award Paper.

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mixing blood serum with thymol buffer is a question which has interested many workers with somewhat conflicting results. Maclagan stated in his paper that this reaction was probably due to an increase in serum gamma globulin. Cohen and Thompson[3] reported electrophoretic analyses in which serum treated with thymol buffer showed a decrease of the beta globulin fraction after the removal of the thymol precipitate. This electrophoretic study indicated that thymol buffer reacts chiefly, if not exclusively, with the beta globulin fraction of serum. The known complexity of the beta globulin fraction in disease makes any attempt at a discussion of the possible nature of a change in this fraction most difficult. It is possible that there is no change in the components of the proteins making up this fraction, but rather a change in the kind and amount of phospholipid chemically bound with the protein. Thus, the exact mechanism of the thymol turbidity test is still unknown.

Experimental Procedure

An investigation was conducted to determine the effect that the age of the serum specimen would have on the results of the thymol turbidity test. Observations were conducted on a series of fifty serums. Fasting blood samples were drawn and thymol turbidity values were done the same day without refrigeration. The serums were then placed in a refrigerator at 4° C. and thymol turbidity evaluations were done the second, fourth, seventh and fourteenth days after the blood was drawn. The results are shown in Table I.

Allowing a slight deviation for technical error, most of the serums tested showed constant values, although there were exceptions. One serum with an initial high value showed a consistent decrease. Four other serums showed a moderate to marked variation. However, the greatest difference between mean levels was only 0.75. At the present time there does not seem to be an explanation for these changes observed.

Another problem studied is the effect that the ingestion of food has on the results of thymol turbidity tests. Mateer[4] and coworkers reported the results of thymol turbidity tests conducted on the blood serums of forty healthy young people. These individuals had no history of illness that could possibly cause liver damage. About half of these bloods were drawn one to two hours after breakfast and the other half one to two hours after lunch. No false positive results were obtained, even though they were taken shortly after eating. A large group of normal individuals was not available to this laboratory, but a series of forty patients being screened for liver damage, or patients with indications of hepatic deficiency, were used for the test. Ambulant patients were selected for this series of tests, as it was felt they would

Table I

THYMOL TURBIDITY VALUES OF FRESH AND AGED SERUM SAMPLES

No.	Day Drawn Units	2nd Day Units	4th Day Units	7th Day Units	14th Day Units
1	12.6	13.6	12.6	12.6	14.6
2	8.8	7.8	7.8	7.1	7.4
3	4.1	5.5	3.6	3.6	4.1
4	5.5	5.5	4.1	4.0	3.3
5	45.0	40.0	35.0	31.2	30.1
6	4.1	5.5	6.0	5.8	4.1
7	4.1	5.3	3.7	3.3	3.3
8	4.0	3.6	3.6	3.3	4.6
9	4.1	4.6	4.1	3.6	6.2
10	3.6	4.1	3.6	3.3	4.1
10	3.0	3.3	3.2	3.0	3.0
12	3.6	3.7	3.3	3.3	4.1
13	5.1	5.5	4.1	5.1	7.4
14	3.1	3.1	1.8	2.1	2.1
15	2.1	3.0	3.3	3.3	4.6
16	8.3	7.8	7.0	7.8	9.8
17	2.1	1.8	1.1	1.1	1.1
18	7.8	7.2	6.6	7.0	7.4
19	3.6	3.0	3.0	3.6	3.6
20	21.2	18.8	18.8	18.8	18.8
21	11.8	10.4	8.8	9.3	8.8
22	8.3	6.8	5.8	6.2	5.5
23	7.8	6.6	5.5	7.0	7.0
24	9.3	10.4	9.8	11.2	10.4
25	29.0	34.6	33.0	45.2	49.2
26	3.0	7.0	3.3	3.3	8.3
27	2.1	2.1	2.1	2.4	2.1
28	20.4	16.7	11.8	17.3	21.8
29	9.3	5.8	5.8	6.6	5.5
30	8.8	8.3	7.8	10.0	10.0
31	3.6	2.6	8.3	1.8	2.6
32	7.8	6.6	7.4	6.6	6.6
33	3.6	3.6	4.1	6.0	5.1
34	8.8	8.8	8.3	6.2	7.8
35	9.8	8.8	7.0	5.8	5.5
36	8.3	7.8	7.0	5.5	5.1
37	4.1	3.6	3.3	2.1	3.0
38	2.8	2.8	3.3	2.6	2.6
39	15.2	15.2	14.1	15.7	14.6
40	8.8	8.3	7.8	7.4	7.0
41	7.8	6.2	5.8	5.1	4.6
42	3.0	2.1	2.6	3.0	3.0
43	4.1	7.0	6.2	6.6	7.6
44	19.6	20.8	20.8	21.2	16.3
45	3.0	3.3	3.6	3.0	2.6
46	3.6	3.3	3.3	3.6	3.3
47	4.1	4.6	4.6	4.6	4.1
48	16.2	16.2	15.2	15.7	17.3
49	9.3	10.4	10.0	12.6	10.8
50	3.0	3.3	2.1	3.0	3.6
Mean	8.25	8.13	7.50	7.81	8.09

take food better than bed patients. A record of diets was made, but this did not prove to be a related factor. Blood samples were drawn before breakfast and again between one and two hours after the noon meal. The day the blood was taken, thymol turbidity tests were done on each of the two serums employing the technique previously described. Results are shown in Table II.

Of the forty pairs of determinations observed, eight showed no change with the ingestion of food, twelve showed only a slight increase that could be within the range of technical error, nine showed a very definite increase, and eleven showed an increase

of one hundred per cent or more. Serums that had a definite chylous appearance after eating exhibited the highest degree of change.

Table II

THYMOL TURBIDITY VALUES OF SERUMS DRAWN BEFORE AND AFTER EATING

No.	Fasting Units	After Eating Units	No.	Fasting Units	After Eating Units
1	3.6	14.6	21	10.4	16.7
2	3.0	8.3	22	10.4	13.6
2	2.6	3.6	23	3.2	3.6
4	3.0	3.0	24	4.3	5.0
5	13.1	13.1	25	1.4	1.4
6	10.0	11.2	26	3.2	4.0
7	4.4	5.8	27	3.6	4.8
8	7.2	12.6	28	2.4	2.4
9	4.6	5.8	29	4.3	8.6
10	5.8	14.1	30	2.5	2.5
11	3.6	8.3	31	1.4	2.4
12	3.0	9.3	32	5.7	6.2
13	5.8	9.8	33	1.2	1.2
14	4.6	6.2	34	1.7	3.6
15	4.6	4.6	35	4.3	11.4
16	5.1	6.4	36	7.2	17.8
17	5.1	5.7	37	7.8	7.8
18	3.0	6.2	38	5.9	8.2
19	5.1	8.3	39	6.8	10.1
20	3.0	12.6	40	5.1	5.7
			Mean	4.85	7.66

The effect of extraction of lipids on thymol turbidity results was observed. Ten of the "after eating" serums were treated with petroleum-ether in an attempt to extract the lipids or fats that had entered the blood serum with the ingestion of food. About equal parts of serum and petroleum-ether were placed in a centrifuge tube. The tube was tightly stoppered and shaken horizontally for five minutes in a Kahn shaker. The excess petroleum-ether and the semi-solid layer underneath were removed with suction through a tube with a wide opening. If the serum was

Table III

COMPARISON OF THYMOL TURBIDITY VALUES OF POSTPRANDIAL SERUM SAMPLES BEFORE AND AFTER EXTRACTION WITH PETROLEUM-ETHER WITH FASTING LEVELS

No.	Fasting Samples Units	POSTPRANDIAL SAMPLES	
		Before Extraction Units	After Extraction Units
1	3.0	9.3	6.2
2	3.0	6.2	4.1
3	3.6	8.3	6.2
4	4.6	10.4	6.2
5	5.8	14.1	9.3
6	5.8	9.8	5.8
7	5.9	8.2	5.9
8	7.2	12.6	11.6
9	10.4	16.7	12.6
10	10.4	13.6	10.4
Mean	5.97	10.92	7.83

still cloudy the above procedure was repeated until clear serum was obtained. Thymol turbidity evaluations were done on the serum. The results are shown in Table III.

Of the ten serums from which the lipids were extracted, all showed lowered thymol turbidity values; three were brought back to the fasting level. Some of the serums seemed to be more resistant than others to this type of extraction. The explanation for this varied resistance is unknown, but it may be that the extractions were incomplete in the seven serums that revealed lowered values which were still higher than the fasting levels. Though the beta globulin fraction is known to be lipid containing, Cohen, and Thompson[5] reported that the total lipid content of serum did not appear to be related to thymol turbidity values. They described a case of lipoid nephrosis with a serum cholesterol content of 1,400 mg. per cent and a normal thymol turbidity value. In a case of lipemia at this hospital, it was noticed that the thymol turbidity values increased while the total lipids were increased under dietary control. When the total lipids reached 6.77 per cent, which was the highest lipid value in this case, the thymol turbidity value was 70 units in a fasting blood sample. After several extractions, using the petroleum-ether technique, the thymol turbidity result was reduced from 70 to 7 units.

Summary

Experiments are reported in which the effect of storage of the serum samples in the refrigerator and of alimentary lipemia on the results of the thymol turbidity test have been investigated. Serums kept in the refrigerator for as long as two weeks showed no significant changes in thymol turbidity values. Because there were some exceptions, however, it is advisable that the test be done on fresh blood serum. An increase in lipids in the blood subsequent to eating produced in some cases an elevation not related to liver disease. Blood tested for thymol turbidity should, therefore, be drawn from fasting patients. Petroleum-ether extraction of serum resulted in some reduction of the turbidity obtained with thymol buffer.

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THE DETERMINATION OF RH-HR (CDE-cde) ANTIGENS AND ANTIBODIES*

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The work of Levine and Stetson,[22] Landsteiner and Wiener[19] opened the door to a new era of progress in the study of blood antigens and antibodies. The Rh problem with its application to transfusion and childbirth is of such importance that the medical technologists must have a clear understanding of the principles involved, as well as the techniques employed. Confusion in the choice of methods has arisen because many techniques of determining Rh antigens and antibodies have been devised by the leading workers in the field. Each method has its advantages as well as disadvantages. Like the serologist using the Kline, Kahn, Wasserman, Eagle and other tests for syphilis, the blood bank and immunological workers have found that no one of the tests can completely furnish the desired information.

To understand clearly the Rh problem, a knowledge of the Rh-Hr (CDE-cde) antigens is essential. It must be remembered that the human erythrocyte is quite complex in its composition, containing not one but many antigens. An understanding of these blood factors is important because an individual may be immunized against any of these antigens which he does not have. The known antigens of the red cell include not only those of the ABO system, the Rh-Hr antigens, P and MN but also the newly discovered S[26], Kell[24], Lewis[23], Lutheran[1], etc. Fortunately, immunization to the rare Rh subgroups (C^w [1], C^v [25], D^u [27], d^{11}, e^{28}), M, N, S, etc. takes place so seldom, that at the present time they have little clinical importance (Figure 1).

The nomenclature of the Rh antigens has been complicated by the use of two systems of classification. Although this problem may be solved in time, it is not the intention of this paper to enter into the merits or demerits of either system. However, from this point the CDE-cde nomenclature of Fisher and Race[9] will be used, since it seems to describe the antigenic components a little more completely and clearly. The translation from one system to the other is not too difficult. This is shown in Figure 2.

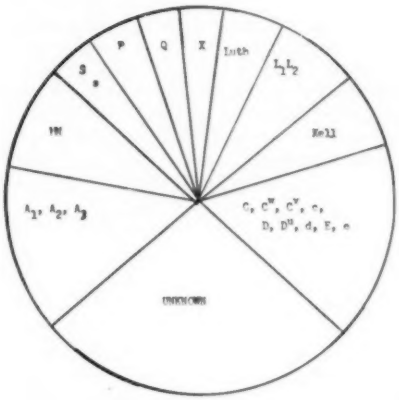
In choosing a method and a serum for the Rh typing test, one must be sure that the serum is suited to the test employed. Most Rh antisera are prepared from the blood of immunized persons

"I would like to express my appreciation to Dr. J. M. Hill and Dr. Sol Haberman of the William Buchanan Blood Center for their advice and encouragement without which this paper would not have been prepared."

* Honorable Mention. Read before A.S.M.T. Convention, June, 1949, Roanoke, Virginia.

FIGURE 1

ANTIGENIC COMPONENTS OF HUMAN ERYTHROCYTES



Graphic representation of some of the known antigenic components of human erythrocytes. The size of the segment does not denote the amount of antigen present.

who may produce one or more of the antibodies which are characterized serologically in Figure 3. The serum for Rh typing should be used exactly as the manufacturer suggests since it has been standardized for the particular method specified on the label.

Rh Typing Techniques

Test Tube Methods:

The test tube method of Rh typing as described by Levine and Stetson[22] makes use of an old serological procedure. It consists of placing one drop of the anti-Rh typing serum in a small tube (7 mm. in diameter and 8 cm. in length) and adding a drop of a 2% red cell suspension from the blood to be tested. The test is placed in a 37° C. water bath for one hour and then centrifuged at low speed, 500-1000 r.p.m., for one minute. Then the sediment is agitated gently and examined under low power of the microscope for clumping. This can be done by placing the tube in a horizontal position on the microscope stage. Best results are obtained by removing the lower lens of the low power objective.

The technique of Landsteiner and Wiener[19, 30] is somewhat similar. It consists of adding two drops of antiserum and one drop of 2% red cell suspension in the same size tubes. The tube

is then incubated at room temperature for one hour. Negative bloods show a circular smooth deposit when examined with a hand lens, while Rh positive bloods show a wrinkled edge or a granular deposit.

The saline tube techniques described above are satisfactory when used with a serum containing saline agglutinins. However, not all Rh antisera can be used by these methods since they may contain the true blocking[28] and/or cryptagglutinoid[17] antibodies which do not react in saline but are detectable by the albumin[8] or serum[29] dilution method.

Figure 2
COMPARISON OF NOMENCLATURE OF Rh-Hr (CDE-ede) ANTIGENS AND SOME COMBINATIONS

Fisher-Race	Wiener	Fisher-Race	Wiener
C	rh′	CD	Rh_o' (R_1)
D	Rh_o	DE	Rh_o'' (R_2)
E	rh″	CDE	Rh_z (Rh_o''')
c	Hr′	CdE	rhy (rh)
d	Hr_o	C″	rh′ ″
e	Hr″		

The very nature of the saline tube technique makes the test valuable when used with a serum which has a saline agglutinin against one antigen and a cryptagglutinoid against a second antigen.[11] For example, one of the serum donors at the William Buchanan Blood Center has a pure saline agglutinin against the "C" antigen, and blockers and cryptagglutinoids against the D antigen.[18] Since there is no saline agglutinin against the D antigen, the serum can be used to detect C by the test tube technique.

Occasionally, false-positive readings in the saline tube test may be due to clumping from excessive centrifugation. The experienced technician can differentiate between the smooth round aggregates of "centrifuge agglutinin" and the rough irregular clumps of true agglutination. These tests must be handled very gently. Rough shaking of the tube before reading, can reduce a strong 4+ reaction to a weak + or a questionable ± reading. This applies to the handling of cross-match tests as well as to Rh typing.

The Diamond-Abelson Slide Test

In this test,[7] the serum should be used full strength. Dilution with 20% bovine albumin or normal AB serum is permissible. One drop of whole blood or a 50% erythrocyte suspension is added to two drops of antiserum on a slide (the "well" variety of slide is preferable). These authors suggest the use of a special lighted view box upon which the slide may be placed and rocked gently back and forth. Agglutination should take place within 3-5 minutes if the blood is Rh positive.

This test depends for the most part upon the presence of

cryptagglutinoid antibodies.[17] A serum which contains chiefly saline agglutinins gives poor results by this method. In reading the slide test the uninitiated technician may have difficulty distinguishing between rouleaux formation and true agglutination. Heating the test above the temperature of the view box should be avoided since the serum and cell suspension may dry and the interpretation may become difficult.

Figure 3
SEROLOGICAL CHARACTERISTICS OF Rh ANTIBODIES

ANTIBODY DESIGNATION	TESTS USED IN CHARACTERIZING ANTIBODIES					
	Saline Tube Test	Blocking Test	Capillary	Albumin Agglutination	Coomb's Developing Test	Hemolysis
Classical Agglutinin	+	—	+	+	+	+
Blocking Antibody	—	+	—	+ or —	+	+
	—	—	+	+	+	+
Cryptagglutinoid	—	—	—	+	+	+
	—	—	—	—	+	+

The Chown Capillary Test

For the capillary test,[2] a saline diluted serum (at least 1 part saline to 1 part serum) and glass capillary tubes 0.3 to 0.6 mm. in diameter and 7.5 cm. long are used. The capillary tube is dipped into the anti-Rh serum, allowing a column of about 2 cm. of serum to enter the tube by capillary attraction. The charged capillary, held in a perpendicular position to avoid bubbles, is then dipped into the blood sample, allowing about the same amount of blood as serum to enter the capillary. The capillary tube is inverted to permit mixing and then placed at a 45° angle in a white rack with a plastacine base. The reaction usually takes place within two to three minutes, and should be considered negative if no reaction has taken place within 10 minutes. The positive test shows beaded threads or bands of agglutinated red cells. The negative reaction shows a long fine smooth thread of sedimented red cells.

The capillary technique has been used extensively as a routine procedure at the William Buchanan Blood Center. A comparative study of 1000 tests using various methods showed the capillary test to have the same specificity and reactivity as the other tests. The simplicity and economy of the test in terms of time and serum has made this technique the method of choice at this Center. No centrifuge, water bath or view box is needed. It was found that 1 cc. of serum is adequate for 60-70 tests in the hands of an experienced worker.

Although the capillary activity of an immune serum seems

to depend chiefly upon the presence of saline agglutinins, this method can be used with more sera than the saline tube technique. The blocking antibody which may mask the saline agglutinin in the test tube has little or no effect in the capillary reaction.

Not all anti-Rh sera are adaptable to the capillary technique. For example, the pure cryptagglutinoid antisera may have no capillary activity and not all saline agglutinin containing sera have satisfactory capillary avidity for use as a test serum. Some undiluted anti-Rh sera give false-positive results by this method; however, the serum may be diluted with one part saline to avoid such reactions. The capillary technique should not be used as a screening test for antibody detection in cases of possible immunization since such a procedure does not detect all antibodies. For example, the cryptagglutinoid and blocking antibodies have little or no capillary activity.

The saline tube method, the slide test and the capillary technique are all good tests when properly used. On occasions the technologist may need to use all three techniques, particularly in the performance of the Rh-Hr genotyping with the rarer subgrouping sera. Unfortunately, these subgrouping sera are not plentiful and serum of this type may be usable by only one method and not others.

CDE/cde (Rh-Hr) Subgrouping

It was first thought that Rh was one antigen inherited by simple Mendelian dominance with a recessive lack of that antigen. Discovery of Rh antisera of different specificity along with the original Hr serum[21] with its antithetical relationship to one of the Rh antisera,[20] led Fisher[9] to predict the six Rh-Hr antigens. These antigens are inherited as a pair of three linked genes on the same chromosome. One set of three is inherited from the father and the other three from the mother. A person may have as many as six or as few as three of the antigens—C and/or c, D and/or d, E and/or e (CDe/cDE, cde/cde).[18]

In the handling of transfusion difficulties and obstetrical immunizations, a knowledge of genotype estimation to determine homozygosity and heterozygosity is necessary.[13] If an individual is homozygous, it means that he has the same antigen on both sides of the chromosome. If he is heterozygous to one of the antigens he has inherited the Rh antigen from one parent and the Hr antigen from the other. It must be remembered that the individual can become immunized against any antigen that is not posseessed.

In advising immunized Rh negative mothers on future pregnancies it is essential that the physician knows whether or not the husband is heterozygous. On the basis of our knowledge of inheritance of the Rh factors, it is known that a homozygous

Rh positive man can father Rh positive children only while the heterozygous man has a 50-50 chance of fathering either Rh positive or Rh negative children.

Since the D antigen seems to be more antigenic and occurs more frequently than the other members of the Rh group, immunization to D is more commonly seen. For that reason, homozygosity is usually expressed in terms of the D antigen. Due to the scarcity of anti-e (Hr″) and anti-d (Hr_o) sera, genotype estimation must be made on a basis of the four available sera, i.e., anti-C (rh′), anti-D (rh_o) and anti-E (rh″) and anti-c (Hr′). On the basis of the reaction of a blood sample in these four antisera, the "d" and "e" antigens can be predicted with considerable accuracy as was shown in a recent survey of random blood samples of a white population in which 14 different antisera including the rare anti-d and anti-e were used.[11, 13, 15]

If a person reacts to the anti-C serum, it means that he has the C antigen on one or both sides of the chromosome. If he is negative to anti-C, he does not have the C antigen on either side of the chromosome and therefore must be homozygous to c. If he reacts to both, anti-C and anti-c sera, he is heterozygous to the C antigen. If he reacts negatively to anti-C, anti-D and anti-E but positively to anti-c, the person has none of the Rh antigens, and therefore must be homozygous to the Hr antigens, c, d and e. Such a person is the Rh negative individual. The presence of the d and e antigens are estimated on the basis of the gene frequency calculations of Fisher.[9] In Figure 4, there is presented the incidence of the various combinations of CDE-cde antigens.

Figure 4

FREQUENCY OF GENE ARRANGEMENTS ON CHROMOSOMES AND INCIDENCE (ACCORDING TO FISHER'S THEORY)

Percent Positive White Bloods (Specificity)	Antibody Designation	COMMON			RARE				
		CDe 43.6 Percent	cde 37.9 Percent	cDE 12.8 Percent	cDe 3.0 Percent	cdE 1.7 Percent	Cde 0.8 Percent	CDE 0.13 Percent	CdE .005
70	Anti-C	+	—	—	—	—	+	+	(+)
85	Anti-D	+	—	+	+	—	—	+	(—)
30	Anti-E	—	—	+	—	+	—	+	(+)
80	Anti-c	—	+	+	+	+	—	—	(—)
65	Anti-d	—	+	—	—	+	+	—	(+)
96	Anti-e	+	+	—	+	—	+	—	(—)

For example, if a blood shows the following reactions:

C D E c

+ + — +

the genotype may be estimated from Figure 4 on the basis of probability.

Since the blood is positive to the anti-C and anti-D and negative to anti-E, the technologists can estimate the arrangement as CDe on one side of the paired chromosome, because the fre-

quency of such an arrangement is 43.6% as opposed to 0.8% for Cde and 3% for cDe occurrence as single arrangements on each side of the chromosome. For the other side of the chromosome, there is the positive reaction to anti-c which indicates the presence of both C and c. The negative reaction to anti-E removes the possibility of the presence of E and therefore only e is present. Consequently, the estimated genotype may be either CDe/cde or CDe/cDe. On the basis of probability, one would estimate the genotype as being CDe/cde since approximately 33% of the population is of that genotype against 1% occurrence of CDe/cDe. This method is a usable tool in the hands of the clinical pathologist.

Antibody Titration Techniques

Detection of immunization through Rh or other blood antigens by pregnancy or transfusion has become an important part of blood banking and serology. A number of tests have been devised for this purpose. The specificity of each of these techniques depends upon the antigen used. For that reason, it is well to have the permanent staff of a laboratory genotyped in order to have cells of the desired antigenic composition. For performing the test it is desirable to use Type O cells or cells of the same blood group as the patient in order to avoid difficulty with the ABO blood group agglutinins. Type O, CDe/cDE blood is desirable for routine work because it will serve as an indicator for immunization against all the CDE/cde antigens except d. The cells used as an antigen keep well and maintain a high antigenicity for about four days if kept sterile and refrigerated.

Saline Agglutinin Technique:

The saline agglutinin technique consists of preparing serial dilutions of the patient's serum in physiological saline, beginning with undiluted, ½, ¼, etc. Usually 1-512 is a convenient end point giving a ten tube test. One drop of each dilution is transferred to small Kahn-type tubes numbered according to the dilution. One drop of a 2% red cell suspension of the blood with desired antigenicity is added to each tube. The test is incubated for 1 hour at 37° C. and then centrifuged for 1 minute at approximately 1000 r.p.m. The tubes are read for agglutination in the previously described manner. The antibody titer of the patient's serum is the highest dilution of serum in which agglutination appears. This test detects the classical saline agglutinin but does not demonstrate the blocking and crypt-agglutinoid antibodies (Figure 5).

The Blocking Test:

The true blocking technique as described by Wiener[28] is designed to detect the degree of inhibition of agglutination by the specific blocking antibodies. The blocking test is done by the

Figure 5

TITRATIONS OF ANTI-Rh SERUM BY VARIOUS TESTS

SERUM I

Test	1	2	4	8	16	32	64	128	256	512	1000	2000	4000	8000	16000	32000	64000
Saline	4+	4+	4+	4+	4+	3+	2+	+	±	–	–	–	–	–	–	–	–
Block	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Albumin	4+	4+	4+	4+	4+	4+	4+	3+	2+	+	–	–	–	–	–	–	–
Developing	4+	4+	4+	4+	4+	4+	4+	3+	3+	2+	+	+	–	–	–	–	–
Capillary	+	+	+	+	+	+	+	+	+	+	+	–	–	–	–	–	–
Slide	+	+	+	+	+	+	+	+	+	+	+	–	–	–	–	–	–

SERUM II

Test	1	2	4	8	16	32	64	128	256	512	1000	2000	4000	8000	16000	32000	64000
Saline	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Block	4+	4+	4+	4+	4+	+	+	–	–	–	–	–	–	–	–	–	–
Albumin	–	–	–	2+	3+	4+	4+	4+	4+	4+	4+	4+	2+	+	±	–	–
Developing	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+	4+	3+	2+	+	–	–
Capillary	+	+	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Slide	+	+	+	+	+	+	+	+	+	+	–	–	–	–	–	–	–

The slide and capillary tests are recorded as either positive or negative rather than including the speed of the reactions which is more important than the size of aggregates for these tests.

usual saline dilution technique with one additional step. Following the first incubation period for the observance of agglutinin titer, a drop of antiserum) which has a saline agglutinin for the antigen being used) is added to the serum-cell mixture. Under ordinary conditions, the added antiserum would yield a 4 plus agglutination with the cells used. If blocking antibodies are present, this reaction will be inhibited and a negative prozone will result (Figure 5). The blocking titer is the highest dilution at which the reaction of the known antiserum is inhibited. This test detects only the true blocking antibody and does not detect the cryptagglutinoid and saline agglutinins.

Tests for Cryptagglutinoids

The Diamond-Denton Technique:

The Diamond-Denton[8] albumin technique has gained widespread use. The serial dilutions of the patient's serum are prepared in the same manner as described for the saline agglutinin and blocking antibody techniques, except that 20% bovine albumin is used as a diluent instead of saline. The cell suspension to be used is also prepared in 20% albumin. The test is sensitive

and is adequate for the detection of both agglutinins and crypt-agglutinoid antibodies. The blocking antibody is frequently responsible for prozones seen in these titrations. Consequently this method should not be used as a one tube screening technique.

Hattersley Technique:

Hattersley[12] has a technique for circumventing the zoning reaction in albumin by centrifuging the test after 5 minutes incubation at room temperature. This author believes that sera which contain blocking antibodies will give a positive reaction at this point. If the test is negative it is incubated for 1 hour at 37° C. and studied for agglutination. This technique has been found to be satisfactory by other investigators.[6]

Coombs, Mourant and Race Technique:

The Coombs, Mourant and Race[5] "developing" technique makes use of an antihuman globulin serum. The test as used by Hill and Haberman[14] consists of several steps. The titration is done by the usual saline dilution technique and incubated in a 37° C. water bath for one hour. At the end of this time, the cells are washed by adding 0.5 cc. of saline to each tube and centrifuging at 2000 r.p.m. for 1 minute. The supernatent saline is completely removed. This is done three times to remove all traces of serum protein other than that in actual antigen-antibody combination with the red cell. One drop of developing serum is added to the washed cells which are then incubated for a second hour at 37° C. At the end of that time the test is centrifuged at 1000 r.p.m. for 1 minute and read for agglutination. The titer is the last tube showing agglutination.

The developing test has the advantage of detecting agglutinins, blockers, and cryptaglutinoid antibodies irrespective of type. This method does not give a zoning reaction since it is a test for antibodies (immune globulins) attached to the cell. In addition to its use in titrations the developing technique may be used as a one tube screening procedure.

The Coombs' test is very sensitive, but it has some disadvantages such as the difficulty of the serum preparation, the necessity of two incubation periods of 30 minutes to 1 hour each, and finally the care that must be taken in washing the cells to avoid contamination with serum proteins. Furthermore, the technologist should realize that an antihuman precipitin serum such as is used in medico-legal work is not necessarily satisfactory for the developing test. If such a serum is used, it must be checked for sensitivity and specificity. Finally, the test may be spoiled by the use of red cell suspensions which are too concentrated (above 3%) since such a preparation will often yield false negative results.

Diagnostic Tests for Erythroblastosis

The Developing Test on Newborn Baby's Erythrocytes:

The Coombs' developing test may be used in the detection of antibodies adsorbed on the red cells of an erythroblastotis baby.[4,10] A drop of a 2% suspension of the baby's red cells are placed in a Kahn type tube and washed three times with saline in the previously described manner. One drop of developing serum is added to the packed, washed, red cells which are then incubated for 1 hour at 37° C. in the water bath. At the end of this time, the test is centrifuged at 1000 r.p.m. for one minute. Agglutination is considered as a positive test for adsorbed antibodies on the red cells. It has been found[10] that the developing test can be used in a diagnostic manner for the detection of erythroblastosis. The test is negative in cases of physiological jaundice, liver disease and anemias of the newborn other than erythroblastosis fetalis. It has been our experience that this test has proved as diagnostic for erythroblastosis as the accepted serological tests are for syphilis.

Witebsky Slide Method:

Witebsky, Rubin, Engasser and Blum[21] have developed a slide technique for the detection of erythroblastosis. Two large loopfuls, or the equivalent, of packed red cells from the baby are mixed with large drop of normal adult serum on a slide. Adult serum from an individual of the same blood group as the baby or Group AB serum may be used for this purpose. Controls using type O Rh positive cells and cells from the adult serum sample are placed on the slide. The cells and serum are mixed by stirring with a wooden applicator. The slide is rocked back and forth for several minutes. Agglutination of the erythroblastotic baby cells should occur in 5 to 10 minutes. These writers consider albumin and normal plasma unsatisfactory substitutes for the adult serum in this test. Some of the weaker reactions may be difficult to interpret.

Discussion:

In the performance of antigen and antibody tests, there are several considerations that apply to all the tests. For example, no serum can retain its ability to give clear reactions without proper care. When not in use, serums must be stored in the refrigerator, preferably the freezing unit, to inhibit the growth of bacteria. Bacterial growth may produce bacteriogenic agglutinins[5] which will give false-positive reactions, while other bacteria may produce inhibitory substances that interfere with agglutination.

The blood sample to be examined is important. If for some reason there must be a delay in examination, the sample is best collected as a sterile clot. The clot is more likely to withstand

the rough treatment of transportation. The antigenicity of red cells remain at a peak for about 4 days. For this reason, red cell suspensions should not be prepared from blood samples that have been stored for more than this period of time. The common anticoagulants, sodium citrate, oxalate, A.C.D. solution or heparin are satisfactory in the recommended proportions but excesses interfere with the tests. Excess oxalate in particular seems to interfere with the capillary test and may give false-positive reactions.

Glassware used in antigen antibody tests must be meticulously clean. Traces of acid or soap may cause hemolysis or false-agglutination or may, on the other hand, inhibit reaction.

Although physiological saline used as a diluent in the tests is the simplest reagent in the laboratory, it may be a source of error. Distilled water and sodium chloride used in its preparation should be of good purity. Intravenous type saline may be used. Saline should be kept in the refrigerator to avoid bacterial contamination which might interfere with the test.

Figure 1 presents 24 of the known human erythrocyte antigens. Any one of these under the proper conditions may be responsible for the immunization of a mother or a transfusion recipient. Fortunately, sensitization to most of these antigens with the exception of the ABO and the CDE/cde systems is extremely rare. For example, at the Buchanan Center only one case of anti-N iso-immunization was identified in 59,508 transfusions over the period of August 1, 1939, to February 1, 1949. Even this case occurred after the 10th transfusion of whole blood. The recipient was type O cde/cde M. His serum showed anti-CD and anti-N antibodies, in addition to only one case of anti-M volunteer donors for Rh serum production. This occurred after the donor had been stimulated with small amounts of Rh positive blood over a long period of time.

Blood typing for the ABO groups is a familiar procedure, and with the proper use of available Rh typing serum, the technologist should have no more difficulty with Rh determinations than in blood grouping. Even genotype estimation, when necessary, is not difficult if the proper sera and a table of gene frequency is available.

In choosing methods for determining sensitization to the CDE/cde antigens it is necessary to consider the different orders of reactivity of agglutinin and agglutinin-like antibodies. These antibodies have been classified by Hill and Haberman into three groups. Witebsky believes that there are possibly four groups. In our laboratory we use the following classification:

1. The saline agglutinin gives a positive reaction by the saline tube technique, the capillary, albumin, and Coombs tests, while the blocking test is negative.

2. The blocking antibody is negative in saline, may or may not react in a capillary, inhibits agglutination in saline, may cause a prozone in albumin and is positive with the Coombs' developing test.

3. The cryptagglutinoid is negative in saline, does not inhibit agglutination, may or may not give a positive capillary test, gives a positive albumin and Coombs' developing test.

The antigen-antibody tests are many and all give good results when properly performed and interpreted. Each of these tests is designed for a specific purpose which it can adequately fulfill, but it cannot be expected to adapt itself to every situation. In figure 5, the reactions of two sera in the more commonly used tests are shown. Serum I shows a positive reaction in all these tests except the blocking test. This means that this serum has a saline agglutinin and a cryptagglutinoid, but no blocking antibody is present. Serum II is negative in saline and shows a prozone in albumin and is weakly positive in the capillary. This serum gives a strong blocking reaction and Coombs' test even in the lower dilutions. The blocking antibody in Serum II not only interferes with the agglutination test in saline, but also causes a prozone in the albumin titration. Consequently if the albumin method had been used as a one tube screening test, the technician would have assumed that this patient had no antibodies. Thus, if a one tube screening procedure is to be used, the Coombs' developing test is best for this purpose. A combination of tests for antibodies gives more complete information than any one test can give by itself.

From the above discussion, it can be seen that a knowledge of the CDE-cde (Rh-Hr) antigen and antibody reactions is as essential to the technologist as the understanding of the various tests for syphilis is to the serologist.

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EXPERIENCE IN THE USE OF THE VAGINAL SMEAR METHOD OF CANCER DIAGNOSIS

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Six years have passed since Papanicolaou's original publication on the recognition of cancer cells in vaginal smears, a monograph which has already become classical. Four years were spent by several doctors in confirming his work. The Memorial Hospital in New York, Warren and Gates in Boston, and Meigs in Boston, were amongst the first to agree with Papanicolaou that the diagnosis of uterine cancer from vaginal smears could be made, within a limit of error, no greater than in the majority of biological tests. Since then most workers have agreed that the method compared with biopsies carries an error of from 1.5 to 3 percent, although exceptional statistics of 9.1 percent has been reported. However, I believe that this large error was due to incomplete follow up with biopsies. Several months later the same authors reported an error of 7 percent. Reports are now accumulating from all over the United States and Canada, most of them calculating the error in the vicinity of 3 percent or less. (Parret, in California; Pund, Georgia; Meigs, Boston; Ayer, Montreal; Seibels, South Carolina.)

The errors are twofold:

First, the negative smears in the presence of cancer. That is to be expected since we can only find such cells that shed from the surface of the tumor. This may be overcome by repetition of smears in suspicious cases. During the past one year and a half, however, many authors have advocated methods by means of which cells are directly wiped or scraped off the surface of the inner portio, the most common site of cervical cancer being at the junction of inner and outer cervix. By applying a direct cervical swab (Pund, Foote) or spatula scraping (Ayer) the percentage of negative cases has been significantly diminished.

Secondly, positive or highly suspicious smears, which could not be verified by biopsy. This error, if indeed it is an error, is of great practical and theoretical significance. The investigation of the uteri in such cases has opened up a new avenue of approach to the problem of cancer research; a field of investigation that started about two years ago.

In July, 1947, I was given the opportunity to study cancer cell diagnosis at Charlotte Memorial Hospital. After several months of studying many slides I felt that this was the most important phase of laboratory work that I had ever done. Although I was not able to secure daily smears on patients as

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* Read before A.S.M.T. Convention, June, 1949, Roanoke, Virginia.
** The fund for this work was made possible by the Charlotte Memorial Hospital and the American Cancer Society, North Carolina Division.

Dr. Papanicolaou recommended, I did have repetitions on many patients that came every week to the Out-Patient Clinic. Also several doctors in town sent me many known cases of cancer for study. The smears were sent with only a limited history of the patient, name, age, last menstrual date, and often without information concerning the clinical diagnosi. A long and tedious period of training lasted for about a year. There are so many different forms that suspicious cells may take and so many formations that non-malignant cells take that experience in looking at many slides is the best teacher.

Although the test is very simple, accurate technique is imperative for satisfactory results. If the smears are not taken and stained properly the results are misleading.

There are three methods of securing smears:

I. From the vaginal vault with the pipet that Dr. Papanicolaou designed.

II. From the cervix with a cotton swab. The swab is rolled gently over a clean slide which is placed immediately in the alcohol-ether solution.

III. Ayre devised a notched wooden spatula for obtaining cervical smears. With a rotary motion he scraps the endocervix and cervical lips. His published results are the best so far reported from the standpoint of percentage of error.

It is unwise for physicians in general to attempt to stain and interpret their own smears without considerable special training and experience We are indebted to Dr. Ayer of Canada for a technique that is satisfactory for mailing purposes. The smears are fixed in the alcohol-ether mixture for 30 minutes, removed and before drying takes place, a few drops of glycerine are added to the slide which is then covered with a clean slide, ready to be placed in a container for mailing. When the slides are received with glycerine they are simply placed in 95 percent alcohol for about one hour to dissolve the glycerine and the smears are ready for the classical Papanicolaou staining process. Therefore, anyone can send slides to cancer detection clinics that are now being established.

I had been looking forward to the day when I would find an unsuspected cancer. That day came last fall. This case was one among twenty smears left on my desk for routine staining. When I found this one which was so typically malignant I was sure it was from a known case, as the doctors frequently brought in smears on malignant cases for me to study. Therefore, in reporting to the doctor I asked if it was on a known positive case and he said that it was not, but could well be. This is the history:

A fifty-four-year-old woman came to a doctor's office because

she had real "cancerphobia," but did not present any signs of cancer. She had been to a doctor two years before because she had had at one time a discharge that was slightly off color. The doctor could see no evidence of cancer at that time, but a curettment was done and the pathology report was negative. She had no further symptoms until about two years later when she again noticed a discharge slightly off color. At this time she showed again no clinical signs or significant findings of cancer. The doctor sent a Papanicolaou smear to the laboratory and the report was "highly suspicious." We asked for a repeated smear. One month later the patient returned for another examination and the repeated smears were positive. At the same time a biopsy from the cervix was performed and reported as positive for infiltrating cancer. One may well assume that this woman had cancer two years ago.

Since that time we have found four more cases. This dramatically points out what Dr. Pund has demonstrated, namely: "Preinvasive carcinoma (of the cervix) gives rise to no symptoms and cannot be detected by physical examination." He found cancer, including the preinvasive type, to be present in 3.9 percent, a figure much higher than the expected incidence of 1.5 percent. One may wonder whether Dr. Pund did not designate lesions as malignant which others would have regarded as benign. Novak, in fact, raised just this criticism, but Pund's evidence seems convincing. In the first place, the cytology of the preinvasive lesions is identical with that of invasive cancer. In the second place, the cytologic changes in such areas are the same as those often found in the immediate vicinity of invasive cancer. Finally, he has compiled a chart of cases from the literature and his own, which show that approximately 18 percent of the preinvasive cancers develop into invasive growths within from one to eight years. He contrasts these 18 percent to the remaining 82 percent, which did not develop into cancer but had received adequate treatment by cervical amputation, hysterectomy or adequate amount of radium. The total number of these cases (81) is too small to render final proof, but the findings are strongly suggestive. Pund, furthermore, showed that the average age of preinvasive cancer is 36.6 years, of covered invasive cancer 42, and of clinically evidenced invasive cancer 48.6 years, indicating that it takes an average of six years for a preinvasive lesion to become invasive and twelve years to become clinically apparent.

If these figures are correct, a periodic check by cervical smears could eventually result in elimination of invasive cancer. Dr. Foote has raised the question whether such procedures would be practical. If one out of 1,500 women over 35 years of an at-random population has cancer of the uterus, its detection

would cost 500 hours laboratory work of a skilled person (at present about $750). If, however, the investigation were confined to gynecological patients, even though these may not reveal symptoms or signs of cancer, the cost would be reduced to 10 labor hours for each positive diagnosis provided we accept Pund's estimate of 3.3 perent.

The use of the cytologic smear for the recognition of cancer could be compared to the use of the blood smear in the diagnosis of diseases of the blood and blood-forming organs. Patient and repeated search of numerous preparations by trained cytologists is essential to sucess. Infiltrating cancer cannot be cured as readily as preinvasive cancer, but if we are to help fight cancer in the early stages, we must train technicians to help the pathologists in this mighty task. Technicians can distinguish benign and malignant cells, but final evaluation is made by the pathologist. This again may be compared to the routine blood smear. The technician makes thousands of normal differential counts, but when she finds one that deviates from the normal she consults the pathologist. After the diagnosis is made from the Papanicolaou smear the diagnosis must be confirmed by biopsy, as the accepted method.

We have examined approximately 1,500 vaginal smears and have identified cancer in 21 of these. All of these cases were verified by biopsies, or after the removal of the uterus. This percentage of 1.4 is low in comparison with other statistics. The material is composed of cases which were submitted to our laboratory as being suspicious of cancer from a clinical point of view, as well as at-random smears from women who had no complaint referable to the genital tract. Many of these smears were also taken from relatively young women, under the age 35, for the purpose of studying vaginal smears under the influence of the menstrual period. Furthermore, we do not possess a proper follow-up of all the cases examined with Papinicolaou smears. In a relatively large number of smears which were reported as being suspicious the patients have not returned to the physician and neither repeated smears nor biopsies were carried out. We will, therefore, have to refrain from drawing conclusions from the actual percentage of positive smears in our laboratory. They are not comparable to the statistical surveys published elsewhere.

We have also examined a total of 18 bronchial smears with positive results in three of them. The diagnosis of carcinoma of the lung by smears from bronchial aspirations may not be regarded as a reliable method. It has been particularly promoted by Herbert in Philadelphia, at the Boston City Hospital, and at Yale University, and it has been found that the diagnosis of carcinoma from the bronchus has been as much as doubled

(Yale) by means of bronchial smears in comparison to bronchial biopsies alone.

While the main point of this paper is to present cancer cells as found by smear technique, there are other possibilities to be considered. There were some (Kernodle and Cuyler) who were not content with only a diagnosis of negative or positive for malignancy. Analysis and correlation of all smears became an impelling research. They have recently published two papers on the Vaginal Cytology of Post-menopausal Women. It is of interest to us because of earlier confusion with some of these sub-type cells with malignant cells.

There have been incidental findings in the smears such as Donovan bodies, red blood sickling, and the ova of Enterobius Vermicularis (Kernolde and Cuyler). We have found Donovan bodies in our laboratory also.

This new avenue of approach in the diagnosis of cancer is not an "open sesame" to all the problems of the clinician, but can be a valuable aid in eradicating cancer.

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HINTS IN THE CARE OF XENOPUS LAEVIS

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The use of the African clawed toad, Xenopus laevis, for the early diagnosis of pregnancy is now generally accepted and is considered accurate. There are, however, three rather unpleasant tasks connected with its use. These are:

1. Getting rid of the eggs after positive results.
2. Feeding.
3. Cleaning tanks after feeding.

In our laboratory we find these tasks can be made easier by:

1. Adding a little sodium hypochlorite solution to the animal jar or container in which the eggs were deposited. This solution dissolves the gelatinous material around the eggs. As soon as this occurs, the containers can be cleaned without difficulty.

2. Feeding fresh heart as well as liver. The meat is cut in thin (about ⅛ inch in width) slabs which are placed side by side on glass plates. These are placed in the freezing compartment of the refrigerator. When ready to feed the toads, take the frozen

slabs of meat and cut into cubes. The partly frozen cubes of meat may be fed to the toads immediately; however, as a rule it is preferable to allow them to thaw.

3. Connecting the feeding tanks with the sewer. This makes flushing them an easy operation. Metal tanks* with drains may be obtained commercially.

* Jay E. Cook, Baltimore, Md.

PARASITES TRANSMITTED FROM ANIMALS TO MAN

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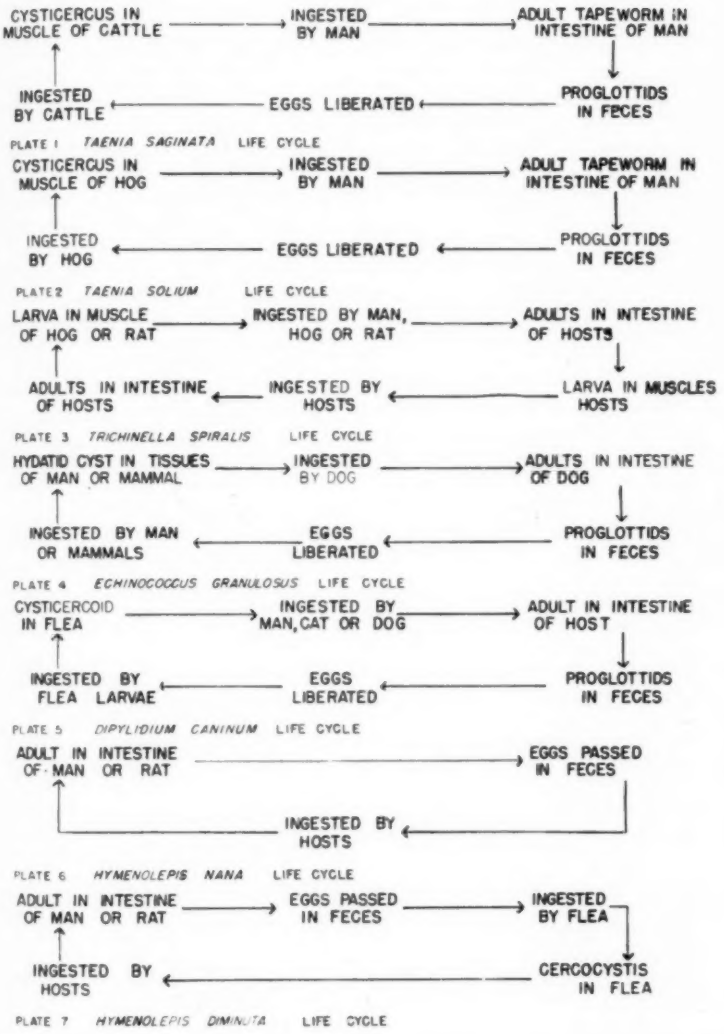
Many parasites found in animals have been reported to be transmitted to man. Of the parasites less commonly transmitted from animals to man the hog serves as a source of human infection for the protozoan ciliate, *Balantidium coli* and the intestinal fluke, *Fasciolopsis buski*. The dog may serve as a reservoir host in certain parts of the world for the protozoan, *Leishmania donovani*, and along with cats as a host for the Chinese liver fluke, *Clonorchis sinensis*. Both cats and dogs in the United States can be hosts for the hookworm, *Ancylostoma braziliense* and the broad fish tapeworm, *Diphyllobathrium latum*. Various animals can serve as hosts for the blood fluke, *Schistosoma japonicum* found in the Far East.

From raw or insufficiently cooked beef one might ingest the larval stages of the beef tapeworm, *Taenia saginata*. By contamination of the hands with eggs from rat feces or ingestion of the larval stage of the dwarf tapeworm in the rat flea, man becomes the host of *Hymenolepis nana*. The dog serves as a source of infection to man for the double-pored dog tapeworm, *Dipylidium caninum*, and the hydatid worm, *Echinococcus granulosus*. The pork tapeworm, *Taenia solium* may be acquired by eating raw or insufficiently cooked pork as may also the round worm, *Trichinella spiralis*.

To understand how these parasites of animals can be transmitted to man a knowledge of the parasite's life cycle is helpful. Man becomes infected with the beef tapeworm by eating raw or insufficiently cooked beef containing the bladder larva stage, a cysticercus (Plate 1). On entering the stomach the invaginated scolex in the cysticercus evaginates, segments or proglottids form and the adult tapeworm develops in the lumen of the intestine of man, the only known definitive host. On passing the ripe or gravid segments filled with eggs or rupturing of the proglottids in the intestine, the eggs are liberated and dispersed in the feces. By eating vegetation contaminated with human feces containing *T. saginata* eggs, the cow becomes infected with the cysticercus, a bladder larva tapeworm stage in the muscles. Prevention of human infection with this beef tapeworm can be accomplished by eating only thoroughly cooked beef, proper disposal of human fecal material and treatment of infected individuals.


CYSTICERCUS IN MUSCLE OF CATTLE
INGESTED BY MAN
ADULT TAPEWORM IN INTESTINE OF MAN
PROGLOTTIDS IN FECES
EGGS LIBERATED
INGESTED BY CATTLE
PLATE 1 TAENIA SAGINATA LIFE CYCLE
CYSTICERCUS IN MUSCLE OF HOG
INGESTED BY MAN
ADULT TAPEWORM IN INTESTINE OF MAN
PROGLOTTIDS IN FECES
EGGS LIBERATED
INGESTED BY HOG
PLATE 2 TAENIA SOLIUM LIFE CYCLE
LARVA IN MUSCLE OF HOG OR RAT
INGESTED BY MAN, HOG OR RAT
ADULTS IN INTESTINE OF HOSTS
LARVA IN MUSCLES HOSTS
INGESTED BY HOSTS
ADULTS IN INTESTINE OF HOSTS
PLATE 3 TRICHINELLA SPIRALIS LIFE CYCLE
HYDATID CYST IN TISSUES OF MAN OR MAMMAL
INGESTED BY DOG
ADULTS IN INTESTINE OF DOG
PROGLOTTIDS IN FECES
EGGS LIBERATED
INGESTED BY MAN OR MAMMALS
PLATE 4 ECHINOCOCCUS GRANULOSUS LIFE CYCLE
CYSTICERCOID IN FLEA
INGESTED BY MAN, CAT OR DOG
ADULT IN INTESTINE OF HOST
PROGLOTTIDS IN FECES
EGGS LIBERATED
INGESTED BY FLEA LARVAE
PLATE 5 DIPYLIDIUM CANINUM LIFE CYCLE
ADULT IN INTESTINE OF MAN OR RAT
EGGS PASSED IN FECES
INGESTED BY HOSTS
PLATE 6 HYMENOLEPIS NANA LIFE CYCLE
ADULT IN INTESTINE OF MAN OR RAT
EGGS PASSED IN FECES
INGESTED BY FLEA
CERCOCYSTIS IN FLEA
INGESTED BY HOSTS
PLATE 7 HYMENOLEPIS DIMINUTA LIFE CYCLE

Taenia solium, the pork tapeworm, has a life cycle (Plate 2) similar to the beef tapeworm except the bladder larva stage, the cysticercus, is usually found in the hog. Man at times has become the

intermediate host through autoinfection and the resulting cysticercus developed in man may be fatal. Thorough cooking of pork, washing of the hands, proper human fecal disposal and treatment of the infected person help to prevent human infections of beef tapeworm.

The only round worm commonly transmitted from hogs to man, *Trichinella spiralis,* has an interesting life cycle (Plate 3) in that man, hog, rat and many other animals are both the definitive and intermediate hosts. Man enters the life cycle by eating raw or insufficiently cooked pork or pork products containing the larvae encysted in the muscles. Due to the action of the digestive juice, the larvae are freed and develop into adults in the intestine of man. These adults being viviparous produce larvae which enter the blood stream and circulate to the muscles where they encyst and later calcify. Hogs become infected by ingesting larvae in infected pork scraps in uncooked garbage or infected rats. This infection known as trichina or trichinosis may be fatal and can be avoided by consuming only thoroughly cooked pork and pork products.

Man can act as the intermediate host for *Echinococcus granulosus* (Plate 4) as the bladder larva stage, the hydated cyst, is found in the liver, lung and other organs of man. Since the adult hydated worm is found in the intestine of the dog, the cestode eggs are passed in the feces. After ingestion of the eggs the hydated cyst develops in the liver, lungs and other organs of man. Preventing the ingestion of the eggs of the hydatid worm would eliminate this infection in man.

The handling of pet dogs and cats provides an opportunity for the accidental ingestion of fleas and lice with the resulting infection of man with the double-pored dog tapeworm, *Dipylidium caninum* (Plate 5). The larval stage of this tapeworm, the cysticercoid, develops in the flea or louse. The adult, double-pored, dog tapeworm develops in the lumen of the intestine of the dog or man. The ova passed out in the dog's feces may cling to the hairs of the perianal region where they are ingested by the arthropod intermediate host.

The life cycle of the dwarf tapeworm, *Hymenolepis nana* (Plate 6) and *H. diminuta* (Plate 7) are very similar except that *H. diminuta* requires an intermediate host to complete its life cycle. In the case of *H. nana* the adult tapeworm is found attached to the intestinal mucosa and the eggs are passed in the feces. The eggs can be ingested by man or rat in which the adult develops or by an intermediate host such as the rat flea where a solid bladder larva stage, the cercocystis, is formed. In the life cycle of *H. diminuta* the eggs must be ingested by an intermediate host in order that the cycle be completed.

All of these life cycles have been indirect, that is, they require an intermediate host with the exception of *H. nana* which may be direct. In all of these infections man is responsible for infecting himself

either by ingesting the egg, raw or insufficiently cooked infected meat or larval form in intermediate host.

In the laboratory diagnosis of parasites it is important to know that material to be examined, the stages that can be expected to be recovered and their chief identifying characteristics. Since we are dealing here mainly with the tapeworms, the adults of which are found in the intestine, the gravid proglottids or eggs are passed out of the body in the feces. The modified zinc sulfate centrifugal flotation method is the method of choice for a concentration of the discussed tapeworm eggs. The procedure is as follows:

1. Using two applicators, prepare a fecal suspension in a Wasserman tube (100 x 13 mm.) by mixing a stool sample about the size of a small pea in half a tube full of tap water. Make certain that comminution is thorough and that all obvious particles are broken up resulting in an even suspension.

2. Add additional tap water until the tube is two thirds full.

3. Centrifuge this preparation for one minute at approximately 2500 r.p.m., first properly balancing the machine.

4. Pour off the supernatant fluid into a container holding a disinfectant, e.g., cresol.

5. Repeat this washing *only* if the stool is extremely oily.

6. Add enough zinc sulfate, sp. gr. 1.20 (approximately 33%) solution to fill the tube half full.

7. Using an applicator, break up the packed sediment very thoroughly.

8. Add additional zinc sulfate solution to fill the tube to within one-half inch of the top.

9. Centrifuge this suspension for one minute at 2500 r.p.m.

10. Without shaking or spilling, carefully place the tube in a rack.

11. Slowly fill the tube brimful with zinc sulfate *without allowing any runover.*

12. Then gently touch a clean, grease-free number 2 coverslip to the meniscus and let stand 2 minutes and gently remove the coverslip with a straight, upward motion.

13. A drop containing helminth ova and protozoan cysts will adhere to the underside of the coverslip.

14. Examine under the microscope for ova and cysts.

A gross examination of the stool may reveal single proglottids or chains of segments. These may appear whitish and have limited movement. The characteristic shape of the proglottid or internal structures seen by staining with Semichon's carmine stain[1] or by clearing in 3-1 carbolic acid-xylol mixture help in the identification. Straining of the entire bowel movement through a series of graded screens may reveal the segments and possibly the scolex. The entire stool sample is thoroughly mixed with tap water so that all the

SUMMARY OF LABORATORY IDENTIFICATION OF PARASITES OF DOMESTIC ANIMALS FOUND IN MAN

PARASITE	Animal Host	Stage in Man	Location in Man	Source of Material	Diagnostic Procedures	Chief Diagnostic Characteristics
Taenia saginata	Cow	Adult tapeworm	Intestine	Feces	Gross examination Screening Direct microscopical $ZnSO_4$ flotation.	Adult: scolex—4 suckers, unarmed; gravid proglottid—15 to 30 lateral uterine branches, one lateral pore per segment. Eggs: 32 to 35u; spherical, hexacanth embryo, thick striated wall.
Taenia solium	Pig	Adult tapeworm	Intestine	Feces	Gross examination Screening Direct microscopical $ZnSO_4$ flotation.	Adult: scolex—4 suckers, 2 rows of 25 to 30 hooklets; gravid proglottid—7 to 12 lateral uterine branches. Eggs: similar to **T. saginata.**
Trichinella spiralis	Pig	Larvae	Muscle	Muscle	Compression Digestion	Larvae—size; 1 mm., coiled in ellipsoidal cysts.
				Blood	Differential	Eosinophilia—20 to 75% suggestive.
Echinococcus granulosus	Dog	Hydatid cyst	Liver Lung	Liver Lung	Examination of cyst at autopsy X-ray	Brood capsule—cysts within cyst containing invaginated scolices. "Hydatid sand"—invaginated scolices in hydatid fluid.
Dipylidium caninum	Dog	Adult tapeworm	Intestine	Feces	Gross examination Screening Direct microscopical $ZnSO_4$ flotation.	Adult: scolex—4 suckers, 1 to 7 rows of 30 to 150 hooklets. proglottids—2 lateral genital pores per segment. Eggs: in clusters surrounded by a membrane, hexacanth embryo.
Hymenolepis nana	Rat	Adult tapeworm	Intestine	Feces	Gross examination Screening Direct microscopical $ZnSO_4$ flotation.	Adult: scolex—4 suckers, 1 row of 20 to 30 hooklets; proglottids—small, trapezoidal; one lateral genital pore per segment. Egg: average 47 x 37u, hexacanth embryo, polar filaments.
Hymenolepis diminuta	Rat	Adult tapeworm	Intestine	Feces	Gross examination Screening Direct microscopical $ZnSO_4$ flotation	Adult: scolex—4 suckers, unarmed; proglottids—similar to **H. nana.** Eggs: average 65 x 75u, hexacanth embryo, no polar filaments.

lumps are removed and screened through graded screens starting with a coarse, then a medium, fine and very fine screen.

In infections with the round worm, *Trichinella spiralis*, an eosinophilia of 20% to 75% is suggestive. An eosinophilia may be present in many helminth and non-parasitic infections. For a positive diagnosis of trichina, a muscle biopsy to demonstrate the encysted larvae is preferred. A thin slice of muscle can be pressed between two glass slides and examined unstained for the encysted larvae. If the larvae are not found, the same piece of muscle can be digested by artificial gastric juice (1% pepsin and 0.5% HCl) incubated at 38° C. for 8 to 15 hours with an occasional shaking. The sediment is examined for free larvae.

The hydatid cyst is best detected by X-ray or Casoni's intradermal skin test.[2] Since the adult hydatid tapeworm is found in animals other than man, the proglottids and eggs are not found in the human stool.

The chief diagnostic characteristics of the scolices, segments and eggs are summarized in Plate 8. All the scolices have four suckers, but may or may not be armed. The mature proglottids all contain at least one set of both male and female reproductive organs per segment. The gravid proglottids are usually differentiated by the number of lateral uterine branches in the case of the genus *Taenia*. The eggs of these tapeworms all contain six hooklets. Each parasite has certain identifying characteristics which when recognized makes their identification easier.

Man acquires most of the parasites of animals through his own carelessness; by ingestion of the egg from fecally contaminated hands, ingestion of the intermediate stages in insufficiently cooked meat, or intermediate hosts.

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A REVIEW OF THE RECOMMENDED NOMENCLATURE FOR CELLS OF THE BLOOD AND BLOOD-FORMING ORGANS*

By MARGRET E. HUGHES**

The nomenclature of hematology has for many years been made up of an accumulation of miscellaneous terms coined by individual investigators from all parts of the world, without benefit of any planned correlation between new terms and existing ones in use elsewhere. Superficial analysis of the terminologies used by different schools of hematology reveals much that is confusing to the teacher and student of hematology. Duplications of terms, inadequate and vague definitions of names and descriptions for cells and diseases are but a few of the factors which have tended to make hematology a seemingly difficult subject to teach, study and understand. Further analysis, however, reveals a lack of planning, for there are many areas where mutual agreement actually exists, although the old terms used do not imply that.

In order to clarify hematologic terminology so that all workers in the field might be able to write and converse on the subject without gross and often unknown misunderstandings and the necessity of restating definitions for all terms used at all times, a group of representative hematologists from all schools, clinical pathologists, anatomists, medical editors and other interested men in related fields got together in a series of "round-table" conferences to discuss the nomenclature of hematology and thereby to determine which were the best of the terms in current use, which terms needed redefining, which should be eliminated entirely and what new terms should be added. The Committee recognized that major differences of opinion still exist regarding the origins, potentialities, functions and interrelationships of certain cells of the blood, and that the terms selected should permit the unequivocal expression of any theory.

Thus, a major step has been taken toward unification and standardization, which by now has become international in scope. Perhaps one of the most important accomplishments has been the arousing of interest in discussing the subject of nomenclature among many groups working in hematology. This interest itself has added much impetus to the project, and has produced many excellent suggestions. The work of this group of authorities is sponsored by the American Society of Clinical Pathologists and the American Medical Association and has

* Presented with the permission of the American Society of Clinical Pathologists.

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received the endorsement of the International Society of Hematology, among others.

The first report[1] of the Committee on Hematologic Terminology, making recommendations for names and definitions of cells of the leukocytic and thrombocytic series, was reviewed before this group at the meeting in St. Paul, Minnesota, in June, 1948; this review[2] was published in the Journal of that Society. Since that time a second report,[3] making recommendations for names and definitions of cells of the erythrocytic series, has been published. In addition, condensations[4,5] of the two reports have appeared in print, presenting in brief form the recommended terminology. Future reports, now in the process of preparation, will give recommendations for the terminology of diseases of the blood and blood-forming organs, as well as of certain cells of the blood-forming organs seen only infrequently in narrow smears or tissue imprints and sections—cells of the histiocytic or reticulo-endothelial system. Reprints of all published reports are available through the American Society of Clinical Pathologists, and to date there has been a wide distribution of them among medical schools, clinical laboratories and other groups who use hematologic terms.

The purpose of the present paper is to continue the presentation of recommended terms which was begun at the St. Paul convention last year, and to reiterate the general purpose and principles guiding the Committee in making its recommendations.

The Committee which has made these recommendations for preferred terminology seeks primarily to attain the clearest, most descriptive and generally *usable* set of terms. It plans to review and revise its recommendations periodically so that nomenclature will not again fall back into the state of confusion from which it is now being lifted. To accomplish this and to maintain a dynamic state in nomenclature, the experiences which are encountered by *all* who study, teach and use hematologic terminology should be made available to the members of the Committee, so that they can best decide which terms are good, and which have not proved adequate when put to the test of usage.

The published reports[1,3] of the Committee contained statements regarding its mode of organization and the procedure by which it functions. "Round-table" conferences were found to be the most satisfactory and efficient means for gathering together and analyzing the different terms in current use, and for discussing and debating the points for and against each. The recommended terms resulted from this type of deliberation, but before final and official approval was given, prepublication reports were sent to *all* members of the Committee, whether or not present at the conferences, to obtain their written suggestions, approval or disapproval of the recommended terms for publication. The terms

as published have received the approval of the majority of the Committee members.

Any of the reports of the Committee may be reproduced by any individual or group, and several professional journals, societies and teaching units have already done so, not only in this country but also in Canada and Australia. The recommended terminology has been adopted into the curriculum of numerous medical schools and other teaching institutions, including such far-flung places as the American University of Beirut, Lebanon, and the University of Santo Tomas in the Philippines.

It should be noted that there has not yet been complete acceptane of the recommended hematologic nomenclature, and that was expected. There are some who feel that either confusion does not exist and there is no need to clarify nomenclature, or the time for it is not now, but perhaps at some indefinite time later on when more exact knowledge has been accumulated. These are certainly sincere criticisms but they are neither constructive nor cognizant of the immediate over-all problem. In Great Britain, particularly, there has been objection to the recommended names for cells of the erythrocytic series, and it would appear that the spokesmen for that country feel that majority opinion favors retaining the "megaloblast-normoblast" designation for the pathologic and normal nucleated erythrocytes. However, the definitions for stages of differentiation which they use are the same as those recommended by the Committee. The point of view of British hematologists is well expressed in an editorial[6] which appeared recently in the journal *Lancet*. In Great Britain, and in most of Europe, these terms have nearly always been used with only one meaning, and uncertainty regarding their exact implication does not exist. That is not true in this country, however, where the term "megaloblast" has been used with several different meanings in schools of hematology with equal standing.

The objections to the recommended terminology which have been expressed so far have been sincere and thoughtful, but unfortunately no one yet has been able to suggest alternative terms which would be accepted by the majority, as were the terms which the Committee recommended.

The Recommended Terms

In reviewing briefly the recommended terms for the leukocytic and thrombocytic series of cells, there will be given only the preferred names, the terms to be avoided and the simple, easily identifiable feature which has been chosen to differentiate the stages of development which are thought to be clinically significant. For complete definitions of terms, you are referred to the original reports[1,3] of the Committee, and to standard textbooks of hematology and histology for the criteria of cell identification.

TABLE 1

Recommended Terms and Terms To Be Avoided When Referring to Cells of a Particular Series or to a Disease Affecting any Cell of that Series

Term To Be Used	Terms To Be Avoided
Lymphocytic	Lymphoid, lymphatic, lymphogenous, lymphocyte, mono-nuclear
Granulocytic	Myeloid, myelogenous, myelocyte, myelocytic, granulocyte, leukocyte, leukocytic, leucocyte, leucocytic
Monocytic	Monocytoid, monocytogenous, mononuclear, monocyte
Plasmacytic	Plasma cellular, plasmacytogenous, myeloma cell, plasmacyte
Erythrocytic	Erythroid, erythrocytoid, erythron, erythrocytogenous, erythrocyte
Thrombocytic	Megakaryocytic, platelet, thrombocyte

In table 1 are given the recommended terms to use and the terms to be avoided when referring in general to cells of a particular series or to a disease affecting any cell of that series. As an example of usage it can be concluded from inspection that the Committee recommends the name "lymphocytic leukemia" rather than "lymphoid, lymphatic, or lymphogenous leukemia." It should be reemphasized at this point that by recommending names for six distinct cell types, mature forms of which can be found in any normal blood smear, the Committee does not imply that it arbitrarily believes each represents a separate line of development all the way back to the most immature or undifferentiated forms. Two or more series may have a precursor cell or "mother tissue" in common, but this theory has not been unequivocally established.

In table 2 are given the stages of differentiation within each cell series. The essenteial morphologic characteristics which distinguished each stage of development from the preceding and following ones are given in italics above and below the arbitrary lines of demarcation, in the left hand column. In all series the most immature form, whether normal or pathologic, is designated as the *blast* stage, and in all instances is characterized by fine chromation structure, with no appreciable area of the nucleus staining darker than any other part. As differentiation proceeds, there occurs a gradual clumping process in the chromatin pattern. In most series it is felt that for clinical purposes only one intermediate stage need be designated at present, and the criterion for distinguishing it is given for each series. In the monocytic series, the intermediate stage—the promonocyte—is distinguished from the monoblast by having an irregularly-shaped nucleus. Like other *pro*-stages it has chromatin structure which has at least one clump, but that is not its most distinguishing feature.

The granulocytic series of cells was fully discussed in the

TABLE 2

Recommended Terms and Terms To Be Avoided When Referring to Specific Cells of the Leukocytic and Thrombocytic Series

Term To Be Used	Terms To Be Avoided
Lymphocytic Series	
Lymphoblast *Fine chromatin structure*	Lymphoid, lymphatic, lymphogenous, lymphocyte, mononuclear Myeloblast, hemocytoblast, lymphoidocyte, stem cell, lymphocyte
Chromatin clump Prolymphocyte *Larger cell size or finer chromatin*	Large lymphocyte, pathologic large lymphocyte, atypical leukocytoid lymphocyte, monocyte, immature lymphocyte
Coarse chromatin structure Lymphocyte	Small, medium or large lymphocyte; normal lymphocyte; small, medium or large mononuclear
Monocytic Series	
Monoblast *Fine chromatin structure*	Monocytoid, monocytogenous, mononuclear, monocyte Myeloblast, hemocytoblast, lymphoidocyte, lymphocyte, stem cell, immature monocyte
Irregularly shaped nucleus Promonocyte *Nucleolus present*	Premonocyte, hemohistioblast, immature monocyte, Ferrata cell
Nucleolus absent Monocyte	Large mononuclear, transitional, clasmatocyte, endothelial leukocyte, histiocyte, resting wandering cell
Granulocytic Series	
Myeloblast *Fine chromatin structure*	Myeloid, myelogenous, myelocyte, myelocytic, granulocyte, leukocyte, leukocytic, leucocyte, leucocytic Granuloblast, hemocytoblast, lymphoidocyte, lymphocyte, stem cell
Chromatin clump Progranulocyte *No specific granules*	Promyelocyte II, leukoblast, myeloblast, premyelocyte, promyelocyte, progranulocyte A
Specific granules Myelocyte *Round or oval nucleus*	Granulocyte, myelocyte B, non-filament, class I
Indented nucleus Metamyelocyte *Bean or kidney shaped nucleus*	Metagranulocyte, juvenile, myelocyte C, non-filament, class I
Part of nucleus with parallel sides Band cell *No filament present*	Staff cell, stab cell, non-filament, class I, rod nuclear, polymorphonuclear, stabkernige, rhabdocyte, non-segmented
Nuclear lobes connected by filaments Segmented cell	Polymorphonuclear, filamented, class II, III, IV or V lobocyte
Plasmacytic Series	
Plasmablast *Fine chromatin structure*	Plasma cellular, plasmacytogenous, myeloma cell, plasmacyte Myeloblast, hemocytoblast, lymphoidocyte, lymphocyte, stem cell, lymphoblastic plasma cell, myeloma cell
Chromatin clump Proplasmacyte *Nucleolus present*	Türk cell, Türk irritation form, lymphoblastic or myeloblastic plasma cell, myeloma cell
Nucleolus absent Plasmacyte	Plasma cell, Unna's plasma cell, Marschalko's plasma cell, plasmacytoid lymphocyte, myeloma cell

TABLE 2—(Cont'd)

Thrombocytic Series Megakaryoblast *Fine chromatin structure*	Megakaryocytic, platelet, thrombocyte Megalokaryoblast
Chromatin clump Promegakaryocyte *Nucleolus present*	Premegalokaryocyte
Nucleolus absent Megakaryocyte *Nucleus present*	Megalokaryocyte
Non-nucleated cytoplasmic fragment Thrombocyte	Platelet, thromboplastid

paper[2] read at the St. Paul meeting. The pleasmacytic series follows the same general pattern, as also does the line of development of the thrombocytic series.

The recommended terminology for cells of the erythrocytic series is given in table 3. After extensive discussion of all current terminologies for this series of cells, the Committee concluded that none of these could be recommended because there was not a majority in favor of any one set of terms used in the past. The term "normoblast" was not liked by many members, who felt that the pattern of using the suffix *-blast* for only the most immature cell of each series should be maintained in the erythrocytic series. In addition, there was not a majority opinion that favored basing the relative degree of maturity on only the progressive acquisition of hemoglobin. It was generally accepted

TABLE 3

Recommended Terms and Terms To Be Avoided When Referring to Specific Cells of the Erythrocytic Series

Term To Be Used	Terms To Be Avoided
Erythrocytic Series Rubriblast *Fine, stippled chromatin*	Erythroid, erythrocytoid, erythron, erythrocytogenous, erythrocyte Erythroblast, megaloblast, pronormoblast, promegaloblast, normoblast, hemocytoblast, stem cell, myeloblast, lymphoidocyte, karyoblast
Chromatin clump Prorubricyte *Nucleolus present*	Erythroblast, megaloblast, pronormoblast, normoblast, macronormoblast macroblast, prokaryocyte
Nucleolus absent Rubricyte *Definite chromatin structure*	Normoblast, pronormoblast, macronormoblast, erythroblast, polychromatophilic normoblast, karyocyte
Pyknotic nucleus Metarubricyte *Nucleus present*	Normoblast, erythroblast, metakaryocyte
Nucleus absent Reticulocyte *Reticulum present with supravital stain*	
No reticulum Erythrocyte	Red blood cell, erythroplastid, normocyte, akaryocyte

that both nuclear differentiation and relative hemoglobin content should be recognized and that this could be accomplished by designating the separate stages according to nuclear differentiation, further characterizing each, if so desired, by means of a qualifying adjective—basophilic, polychromatic or norchromatic —to describe the amount of hemoglobin present. Thus, many deviations from normal can be more exactly characterized by only one set of terms.

The most difficult decision to reach was choosing the stem to describe the nucleated forms of the erythrocytic series. Latin and Greek authorities were consulted and many stem-words were considered. The majority finally decided on the Latin stem "rubri," since it is easy to pronounce, readily understood and has been used in the formation of many other medical terms. It has been criticized by a few, for its use results in hybrid terms when combined with the Greek prefix and suffix *pro-* and *-cyte*. However, hybrid terms are common in our language—for example, "automobile," "autoclave" or "gravimetric"—, and when the advantages inherent in this term are weighed against this one criticism it seems to have only minor significance.

In order to describe the morphologic deviations from normal which characteristically are seen in the nucleated erythrocytes and the granulocytes of marrow from cases of untreated pernicious anemia and other macrocytic anemias which respond to therapy with liver extract, folic acid or vitamin B_{12}, the Committee decided to recommend the use of a descriptive, qualifying adjective phrase, which would apply equally well to the cells of both the erythrocytic and granulocytic series and to the blood and marrow pictures as a whole. They chose the expression "pernicious anemia type," and recommend its use in connection with the name of the cell being described. Thus, one would say "pernicious anemia type polychromatic prorubricyte" to indicate a cell whose nuclear structure was only just beginning to show clumping of the chromatin, which still contained a nucleolus, whose nuclear pattern had a more open network structure—indicating a relative increase of parachromatin and decrease of basichromatin as compared to normal—and whose cytoplasm showed from its staining characteristics the presence of hemoglobin. One might also speak of a "pernicious anemia type neutrophilic metamyelocyte" to describe one of the bizarre shaped and often extremely large cells which are equally characteristic, and which also revert to normal after adequate therapy with anti-pernicious anemia principle.

The essential features which characterize the stages of differentiation within the erythrocytic series are fundamentally much the same as they are for other cell series, based on what is thought to be the basic process of maturation for all cells of any

tissue. The most undifferentiated cell of the series is the *rubriblast,* having very fine chromatin structure, frequently in a dotted or stippled pattern, rather than lace-net-like. Nucleoli are usually discernible. The next stage is the *prorubricyte,* having a coarsened nuclear structure, but still containing nucleoli. The *rubricyte* is the third significant stage, characterized by loss of nucleoli and a nucleus which, though markedly clumped, still has a recognizable chromatin pattern. In the *metarubricyte* stage of development the nucleus has lost all structure, is dense and solid-appearing, and is described as being pyknotic. It may be fragmented, partially autolyzed or partially extruded, but it is still recognizable as being of nuclear origin. The end stage of this series is the *erythrocyte,* a non-nucleated cell which needs no further description. The *reticulocyte* is thought to be a young erythrocyte; it can be distinguished only in supravitally stained preparations.

The foregoing is only a summary of the recommended names for cells of the trythrocytic series. It must be clearly recognized by all that the presently recommended names in all series are not thought to be perfect in all respects. However, they do have a great advantage in teaching, for they permit the teacher to say broadly that the most undifferentiated cell in all series is the *-blast;* the second stage is the *pro-;* the third stage is the *-cyte;* the fourth stage is the *meta-;* and the most differentiated, end stage carries the suffix *-cyte,* and in all but one instance is the term used to describe the series as a whole—thus, lymphocyte, lymphocytic; thrombocyte, thrombocytic.

It has been suggested by the Committee that everyone put these terms into immediate and continued use, for their success or failure depends on their being given an extensive and fair trial period by all. If an unbiased, critical appraisal—on a national or international scale—of the recommended nomenclature can be achieved, then indicated revisions and additions can be made in a planned, orderly manner. All medical technologists, as well as their clinical pathologists and associated physicians and surgeons are urged by the Committee on Hematologic Terminology to use these terms and to note especially any significant cells or diseases which have not been given consideration by the Committee. As knowledge in hematology increases it is expected that additions in terminology will have to be made. These should be brought to the attention of the Committee. An Interim Committee, to receive and review all suggestions, has been appointed and consists of the following: Dr. Frank H. Bethell, Chairman, Ann Arbor, Michigan; Dr. Frank J. Heck, Rochester, Minnesota; Dr. K. J. R. Wightman, Toronto, Canada; Dr. C. J. C. Britton, London, England; and Dr. L. J. Witts, Oxford, England. Present plans call for a revision of the nomenclature every five or ten years, for it takes at least that long in the field of science to gain a true evaluation of anything new.

Summary

The terms recommended for cells of the leukocytic, thrombocytic and erythrocytic series are briefly reviewed and the guiding principles and future reports of the committee for clarification of the nomenclature of cells and diseases of the blood and blood-forming organs are described. It is suggested that all who use hemotologic terminology put the recommended nomenclature into immediate use, so that it can be given an adequate trial period.

REFERENCES

1. ————————: First report of the committee for clarification of the nomenclature of cells and diseases of the blood and blood-forming organs, Am. J. Clin. Path., 18:443-450, May, 1948.
2. Hughes, Margret E.: An explanation of the terminology and definitions recommended by the committee for clarification of the nomenclature of cells and diseases of the blood and blood-forming organs, Am. J. Med. Tech., 14:324-336, Nov., 1948.
3. ————————: Second report of the committee for clarification of the nomenclature of cells and diseases of the blood and blood-forming organs, Am. J. Clin. Path., 19:56-60, Jan., 1949.
4. ————————: Condensation of the first two reports of the committee for clarification of the nomenclature of cells and diseases of the blood and blood-forming organs, Blood, 4:89-96, Jan., 1949.
5. ————————: Recommended terms and definitions for cells of the leukocytic, erythrocytic and thrombocytic series, J. A. M. A., 139: 175-176, Jan. 15, 1949.
6. Editorial: Names for blood cells, Lancet, 1:486-487, March 19, 1949.

ABSTRACTS

A SINGLE SOLUTION IRON-HEMATOXYLIN STAIN FOR INTESTINAL PROTOZOA. By Morris Goldman, from the U. S. Public Health Service Communicable Disease Center, Laboratory Division, Atlanta, Georgia. Stain Technol. 24:57, (Jan.) 1949.

A single solution iron-hematoxylin stain is described for rapid staining of fecal smears.

Methods (Quoted verbatim)

"Two solutions are used in preparing this stain:

SOLUTION A:

A 1% solution of hematoxylin in 95% alcohol; prepared by diluting a stock solution of 10% hematoxylin in 95% alcohol. This solution need not be ripened.

SOLUTION B:

Ferric ammonium sulfate-violet crystals (iron alum).	4.0 g
Glacial acetic acid	1.0 ml
Conc. sulfuric acid (sp. gr. 1.8)	0.12 ml
Distilled water	100.00 ml

For use, mix equal parts of Solution A and Solution B. The mixture turns a rich purple and overstains when first prepared. Within a few hours the color changes to a purplish brown. At this point the solution should be filtered and it is then ready for use. If the solution becomes greenish-black, it is unsuitable for staining purposes. Use a freshly prepared solution if this blackening has occured.

"The following staining schedule is recommended:

(1) Fix smears in Schaudinn's plus 5% acetic acid for 5 minutes at 50°-60° C., or for one hour or more at room temperature.

(2) Wash in iodized 70% alcohol for 5 minutes.

(3) Transfer through two changes of 50% alcohol for 3 minutes each.

(4) Stain in iron-hematoxylin (A & B mixture) for three minutes—some specimens require different timing—this is the average.

(5) Wash in running water for at least 5 minutes. (15-30 Minutes for permanent mounts.)

(6) Dehydrate and clear through alcohols, carbol-xylene, and xylene, about 5 minutes in each.

(7) Mount in Clarite or other neutral mounting medium."

According to the author this stain lasts about a week in open Coplin jars. Fresh solutions stain smears a bright blue—as the stain becomes older the stains give progressively grayer stains. Nuclei, chromatoid bars, fibrils, and other morphological details are usually well defined in the majority of specimens stained with this modification.

STAINING THE TUBERCLE ORGANISM IN SPUTUM SMEARS. By Mary A. Darrow, Biological Stain Commission, Division of Biochemistry, University of Rochester Medical School, Rochester, N. Y. Stain Technol. 24:93, (April) 1949.

In an investigation carried out to evaluate the accuracy of cold staining Tubercle organisms, filtered Kinyoun's, the usual Ziehl-Neelson formula, and a third formula intermediate in strength between these two were used. No appreciable difference between the three solutions was noted. The cold staining method is simpler, quicker, and when staining jars are employed close watching is unnecessary.

Technic for Cold Staining

Cold staining fluid..... 3-5 minutes
Rinse in tap water
3% acid alcohol (70-95%)..... 15 seconds to 2 minutes
Wash in tap water
Stain 1 minute with 1% aqueous methylene blue
Wash in tap water

In the experience of the writer it has not been necessary to check her findings by the conventional hot-staining method.

THE DETERMINATION OF TRUE GLUCOSE IN BLOOD BY REDUCTION OF FERRICYANIDE: The Production of a Linear, Reproducible Color Without the Use of a Stabilizing Agent in Macro- and Microtechniques Suitable for Visual and Photoelectric Colorimetry. By George R. Kingsley, M.S., Clinical Biochemistry Laboratory, GM&S Hospital, Veteran's Administration Center, Los Angeles, Calif., and John G. Rheinhold, Ph.D., Division of Biochemistry, The Laboratories, Philadelphia General Hospital, Philadelphia, Pa. Jour. Lab. and Clin. Med. 34:713 (May), 1949.

In this paper, a sensitive method yielding a color of sufficient stability and reproducibility for measurement in a photoelectric colorimeter without the need for frequent recourse to standard solutions and giving precise results over a wide range of true blood sugar values is described. The method is applicable to protein-free filtrates of both low and high dilutions. (1-10 and 1-100).

Macromethod. (Quoted verbatim)

REAGENTS: Chemicals used are of analytical reagent quality unless otherwise noted.

Alkaline Potassium Ferricyanide: Weigh exactly 1.84 Gm. of anhydrous sodium carbonate and 0.8 Gm. of potassium ferricyanide and dissolve together in 1 liter of distilled water. Keep in dark bottle away from light.

Ferric Iron: Dissolve exactly 2.0 Gm of ferric ammonium sulfate ($Fe(NH_4)(SO_4)_2 \cdot 12\ H_2O$) in 945 ml water and add 55 ml of 85% orthophosphoric acid.

Acid Cadmium Sulfate: 13.0 Gm cadmium sulfate ($3CdSO_4 \cdot 8$

H_2O) or 10.59 Gm of anhydrous cadmium sulfate and 63.5 ml of exactly 1 N sulfuric acid are brought to a volume of 1 liter with distilled water.

1.1 N Sodium Hydroxide.

Stock Glucose Standard: 1.000 Gm made up to exactly 100 ml with 0.25% benzoic acid.

STANDARDIZATION: 0.5., 1.0., 1.5., 2.00., 2.5., 3.0., and 4.0., ml. of stock glucose standard are each diluted to 100 ml. 0.5 ml of these standards is treated in the same manner as 0.5 ml of blood filtrate as outlined under procedure. Plot readings against concentration of the glucose standards or use a conversion factor: Mg% glucose K (2-log%T) If blank readings above 8 on a Klett-Summerson or 0.03 with Coleman spectrophotometers are obtained on the scale readings, the reagents are not suitable for use. (K equals 335 for Coleman spectrophotometers using Models 6 & 14 and 6-304B cuvettes).

PROCEDURE: To 8 ml/of acid cadmium sulfate solution add 1 ml of whole blood, mix thoroughly, let stand one minute, then add 1 ml of 1.1 N sodium hydroxide and mix by shaking. Allow to stand for 15 minutes; filter through Whatman #40 filter paper, or preferably centrifuge at 3,000 rpm 5-10 minutes."

Place 0.5 ml of filtrate into a test tube graduated at 20 ml. Then add 5 ml of the alkaline potassium ferric cyanide solution. Mix by twirling; place in a boiling water bath for 10 minutes. Cool in a 40° C water bath for three minutes; add 5 ml of the ferric iron solution and mix by twirling; allow to stand for 5 minutes in the 40° C water bath. Dilute to the 20 ml mark with distilled water and mix. Read in the photoelectric colorimeter within 20 minutes. Use a green filter transmitting light between 500 mu and 570 mu with a maximum transmission at 540 mu.

A more dilute filterate may be prepared by adding 1 ml of whole blood to 18 ml of an acid cadmium sulfate solution prepared as follows: 5.77 Gm cadmium sulfate ($3CdSO_4 \cdot 8\ H_2O$) or 4.7 Gm anhydrous cadmium sulfate ($CdSO_4$) and 28.2 ml of exactly 1 N sulfuric acid made into 1 liter of solution with distilled water. Add 1 ml of 1.1 N Sodium hydroxide one minute after mixing 1 ml of blood with the above described cadmium sulfate solution. Continue procedure as directed.

Micromethod

REAGENTS: *0.1% Stock Glucose Standard.* Dissolve 100 mg glucose and dilute to 100 ml with 0.25% benzoic acid.

0.01% Dilute Stock Glucose Standard: Dilute 10 cc of stock standard to 100 ml with 0.25% benzoic acid.

Alkaline Potassium Ferricyanide: Same as macroreagent.

Ferric Iron: Same as macroreagent.

Dilute Acid Cadmium Sulfate for 0.05 ml Pipette: Dilute 40 ml of reagent used in macromethod to 100 milliliters.

Dilute Acid Cadmium Sulfate for 0.02 ml Pipette: Dilute 36 ml of reagent used in macromethod to 100 milliliters.

0.055 N Sodium Hydroxide for 0.05 ml pipette.

0.022 N Sodium Hydroxide for 0.02 ml pipette.

STANDARDIZATION: Measure 1, 2, 4, 6, 8, 10, 12, 15, and 20 ml of dilute stock glucose standard with a burette and dilute to 100 ml with 0.25% benzoic acid. add 0.5 or 1 ml of these dilutions to colorimeter tubes and treat on the same manner as directed for 0.5 and 1ml of blood filtrate. One ml of 1-100 dilution of 0.01 per cent stock glucose standard is equivalent to 10 mg of blood glucose if the micromethod for 0.02 ml of blood is used, or 8 mg if the micromethod for 0.05 ml of blood is used. Readings may be plotted against the concentration of the standard or a conversion factor may be used as indicated in the macro method. K equals 503 in the micromethod for 0.02 ml pipettes of blood for Coleman spectrophotometers 6 & 14 with 6-304B cuvettes. K is not given for the 0.05 pipette.

PROCEDURE FOR 0.02 ml PIPETTES: Add 0.02 ml of whole blood to 1 ml of dilute acid cadmium sulfate solution for 0.02 ml pipettes in a small Kahn tube and mix by washing out pipette; let stand for one minute. Add 1 ml of 0.022N Sodium Hydroxide, stopper and mix. Let stand for 15 minutes, then centrifuge. Add 1 ml of the clear supernatant filtrate to 1.5 ml of alkaline potassium ferricyanide in a colorimeter tube graduated at 6 ml. Digest 10 minutes in a boiling water bath. Cool 2 minutes in 40° C. water bath; add 1.5 ml. of the ferric iron reagent; mix and allow to stand for 5 minutes in the 40° C. bath. Make a reagent blank by treating 1 ml of distilled water in the same manner as 1 ml of filtrate and set at 100% transmission with light at 540 mu.

PROCEDURE FOR 0.05 ml PIPETTES: Add 0.05 ml of whole blood to 1 ml of the dilute acid cadmium sulfate solution for 0.05 ml pipettes and in a small centrifuge tube and mix by washing out pipette; let stand 1 minute. Add 1 ml of 0.055N Sodium Hydroxide, mix and stopper. Let stand 15 minutes and then centrifuge. Add 0.5 ml of the supernatant filtrate to 1.25 ml of alkaline potassium ferricyanide in a 5 ml graduated Klett colorimeter tube. Digest 10 minutes in boiling water; cool 2 minutes at 40° C. Add 1.25 ml of the ferric iron solution, mix and let stand for 5 minutes. Dilute to the mark and read within 20 minutes as directed in the preceding method.

Twenty-three normal males were used in the various experiments necessary to establish the normal range of true blood sugar by this method; 73-85 mg % were the values obtained.

BOOK REVIEWS

MEDICAL ETYMOLOGY: By O. H. Perry Pepper, M.D., Professor of Medicine, University of Pennsylvania. 263 pages. Philadelphia and London. The W. B. Saunders Company. 1949.

Perhaps the most neglected related subject to any phase of the practice of medicine is etymology—the science of the origin, changes, and inflections of words. In the days when Greek and Latin were incorporated in the standard university curricula, words were not as troublesome for general medical students as they are today. A half century ago Word Analysis was a part of the standard high school course; college graduates were trained in rhetoric; and their vocabularies were precise as a result of such training. As a measure to meet this tremendous gap in our educational system in our present university curricula, Dr. Pepper has compounded this handbook for students of medicine, dentistry and nursing. Although the medical technologist has not been mentioned by the author—the words analyzed in this book will greatly improve any technologist's approach to her work.

To those of us who have assisted in the training of technologists nothing is more exasperating than to have a student remark at the end of a lecture that *she* did not understand a single word in the whole lecture. If a technologist's training is based upon actual technology of the various medical sciences comprising the whole clinical pathology, it is paramount that a certain amount of etymology be included in an introductory course, particularly if that technologist is to be an intelligent worker. Thus it is that the need for this book is so apparent to the reviewer.

The book is divided into four parts: Part I, the introduction, dealing with the background of medical terminology, prefixes, suffixes, compounds, transliteration, eponyms and onomatopoetic words, forms a basis for the remainder of the book; Part II lists those words found to be the most troublesome in the preclinical subjects of anatomy, biochemistry, physiology, pathology, bacteriology, pharmacology and therapeutics and parasitology. Part III is devoted to clinical subjects and Part IV to dentistry.

This book is not in any sense an abridged medical dictionary, the origin and derivation of a word is given rather than its exact meaning in present day usage. Thus the meaning of any new term is usually made clear to the student by its use and context as soon as the word is encountered.

The reviewer wishes to recommend this book to all students of the basic medical sciences and particularly to training schools of medical technology as a little volume containing a wealth of information which, when learned, will serve that student for life.

ZINSSER'S TEXTBOOK OF BACTERIOLOGY: The Application of Bacteriology and Immunology to the Diagnosis, Specific Therapy and Prevention of Infectious Diseases for Students and Practitioners of Medicine and Public Health. Revised by David Smith, M.D., Professor of Bacteriology and Associate Professor of Medicine; Donald S. Martin, M.D., M.P.H., Professor of Preventive Medicine and Public Health and Associate Professor of Bacteriology; Norman F. Conant, Ph.D., Professor of Mycology and Associate Professor of Bacteriology; Joseph W. Beard, M.D., Professor of Surgery in Charge of Experimental Surgery; Grant Taylor, M.D., Associate Professor of Bacteriology and Associate Professor of Pediatrics; Henry I. Kohn, Ph.D., M.D., Surgeon U.S. P.H.S., Assistant Professor of Physiology and Pharmacology (on leave); and Mary A. Poston, M.A., Instructor in Bacteriology, the Duke University School of Medicine, Durham, N. C. Ninth edition. 992 pages, 251 figures. New York: Appleton-Century-Crofts, Inc. 1948.

Hiss and Zinsser's textbook of bacteriology is as classic as bacteriology, having maintained a place second to none in its field and scope. The present revisors need no introduction to the medical field. This new ninth edition is certainly welcomed by those who grew up with the previous editions. Throughout the entire text there are many illustrations well selected and chosen which will be of inestimable value to beginning students in medical bacteriology. The chapters devoted to fungi are well written and direct to the point. The reference material deserves special commendation. It must be realized that it is impossible to include all of bacteriology in less than 1000 pages—yet when the proper references and lists of acceptable texts are included at the end of each chapter, if one has access to a standard medical school library, the wealth of information on the subject is tremendous.

This book can certainly be recommended to universities, colleges, medical schools, and schools of medical technology and nursing either as a text or as a necessary reference. No library for technologists would be complete without it.

TECHNIQUES OF HISTO- AND CYTO-CHEMISTRY. By David Glick, Ph.D., Associate Professor of Physiological Chemistry, The Medical School, University of Minnesota. 531 pages, 159 figures. 1949. New York and London. Interscience Publishers, Inc.

This book is a manual of morphological and quantitative micromethods of inorganic, organic and enzyme constituents of biological materials presented in a truly fascinating manner for scientists. The material is given in detailed form and covers an amazing number of subjects and includes many techniques not readily accessible outside of large medical libraries. Histology and cytology of past eras have now become a new field of histo- and cyto-Chemistry—new technics have been evolved and newer methods and apparatus have supplanted those in use a decade ago. A considerable portion of this newer chemical and physical technology has been incorporated in this timely volume which has appeared somewhat prematurely.

After perusing this book the reviewer is cognizant of the fact that micro-chemistry is becoming of age; it is not just a subject given as graduate credit in the most exclusive universities; it is the newer technology which is capable of bringing our vision into the living units, and to the molecules and atoms.

Research institutions and laboratories will find this a most advanced volume containing much useful information. This book is a very valuable contribution to its respective field; the references are par excellent; nevertheless, it is doubtful if it will prove of much practical value to the clinical laboratory, in that the technology is mostly too advanced for the average technical worker of today.

THE PRESIDENT'S PAGE

Dear A.S.M.T. Members:

I should like to introduce to you the members of our newly created Public Relations Committee. The Chairman is Miss Frieda Claussen of St. Paul, Minnesota, and her associates are Miss Mary Nix, Portland, Oregon; Miss Lavina White, Pueblo, Colorado; Miss Josephine Pyle, Middletown, Connecticut; and Miss Doris Boon, Charleston, West Virginia. To this group we are giving the responsibility of how best to reach a sound and propitious relationship with the public.

We do not ask or expect them to accomplish this alone. I hope that the state societies will follow the action of the American Society of Medical Technologists by adding to their organizations such Public Relations Committees as will work with the national group.

There is more we can do to help the progress of our Public Relations than offer suggestions to this committee. It is our desire to have the public realize that we, as Medical Technologists, are an integral part of Medicine. Our daily conduct as professional people, our attitude toward our profession, the way we practice it, the way in which the public sees us in action, can go a long way toward accomplishing the wholesome relationship we desire. It can do more than any organized effort, although that, too, has its value. However trite this homespun theory may seem, it is a recapitulation of truth, which, after all, is what we scientists are ever seeking.

Sincerely yours,
IDA L. REILLY, MT (ASCP)
President, A.S.M.T.

NEWS AND ANNOUNCEMENTS

MORE NOTES FROM THE 17TH ANNUAL CONVENTION

Roanoke, Virginia, June 19-23, 1949

At the meeting of the House of Delegates, some 179 Board members and delegates answered roll call. The decision of the Board of Directors concerning the request of the Georgia Society of Medical Technologists to have the official insignia of A.S.M.T. placed on the state society letterheads to designate affiliation, was appealed to the House of Delegates. The House approved the use of a seal to designate affiliation, with the design and the method of handling to be left to the discretion of the Board of Directors. After the distribution of mimeographed copies of the reports of the standing committees, the request of the Advisory Council that a Public Relations Committee be appointed, was discussed. It was decided that such a committee be appointed for the coming year, and that it be added to the list of Standing Committees as soon as this could be done through the By-Laws of the Society.

After the election of officers, the following changes were voted into the Constitution and By-Laws: Article VIII, Section 1. (b) add to read: "*Affiliate* members who shall hold a certificate from and be in good standing with the Board of Registry of the American Society of Clinical Pathologists at an educational level lower than that required for the certificate of MT (ASCP). They shall enjoy all the privileges of the society except office-holding and voting." The sub-section defining Honorary Members becomes (c). Sub-section (d) was added to Article XI, Section 4, to read. "For accepting Active or Affiliate members who do not meet the requirements of this Society."

Articles XII and XIII were revised to permit the Board to amend the Constitution and By-Laws in emergencies, subject to the approval of the House of Delegates at its next meeting. (See March A.J.M.T., Vol. 15, No. 2, page 102.) The proposed amendment to Article XIII, which will prevent a minority block of the Board of Directors, also was passed. The By-Laws were amended as follows: Article I, Section 3, add (h), to read, "See that the requirements for Active and Affiliate members agree with the membership requirements of this Society." Articles II and III, Sections 1 & 2 and 3, (of III) to insert after the word, "active," in each case, the words, "and affiliate"; and to insert at the end of this section, "unless the records of the Executive Secretary of this Society indicate that such members are not eligible, and the subordinate society does not furnish proof of their eligibility." And Section 3, to strike out the words: "provided he meets the requirements for membership in the new

subordinate society," also was passed. In Article IV, Code of Ethics, the code was accepted as read except Section I, "Nothing in this Code of Ethics shall be inconsistent with that of the American Society of Clinical Pathologists and that of the American Medical Association," and Section V, "I will not accept work outside the practice of my employer." Section V. was rejected on the basis of the fact that many certified medical technologists necessarily break this section of the Code due to the very nature of their duties, and the circumstances of their employment, and such rejection was a "token" rejection of the identical section of the Registry Code of Ethics. A motion to the effect that this action was to be brought to the attention of the Board of Registry, was also passed. The remaining amendments were accepted.

A resolution protesting the use of the term, "nurse technician" by schools of nursing, for a form of "practical" nurse, was adopted, and a committee was requested for the carrying out of its provisions.

The invitation of the Texas Society of Medical Technologists, to A.S.M.T. for the June 1950 convention to be held in Houston, Texas, first given two years ago, was officially accepted. In 1951, the Society will hold its annual convention in Boston, Massachusetts.

PLAN NOW TO ATTEND THE CONVENTION IN HOUSTON, TEXAS, ON JUNE 11-15, 1950, at THE SHAMROCK.

ANNOUNCEMENTS
NOMINATIONS AND ELECTIONS

Letters have been sent by the Nominations and Elections Committee to the affiliated societies of the A.S.M.T. soliciting suggestions for the slate of officers for the next election. As individual members of the society, the committee would appreciate having your ideas and preferences with the qualifications of prospective candidates sent to your state secretaries, who will forward them to this committee. The suggestions may also be sent direct to the committee members.

This is your opportunity to tell us whom you would like to have as officers of your society. Each suggestion will be given careful consideration. Your cooperation is needed NOW. Please send your suggestions by December 1, 1949.

Offices to be filled: President-elect, Recording Secretary, Board of Directors (two).

NOMINATING COMMITTEE: Henrietta Lyle, Washington County Hospital, Hagerstown, Md., Chairman; Gladys Jacobs, 212 Garfield St., Bay City, Michigan; Hazel Current, 918 - 17th St., Santa Monica, California; Oscar M. Alton, 2036 Tyler Lane, Louisville 5, Kentucky; Mrs. Violetta Wakefield, 1209 North 34th St., Fort Smith, Arkansas; Louise Miller, 28 B Elizabeth Road, Hampton, Virginia.

OTHER STANDING COMMITTEES

CONSTITUTION AND BY-LAWS: L. B. Soucy, Chairman, 204 S.E. Fifth St., Plainview, Texas; Sylvia Anderson, Wisconsin; Allyne Lawless, Colorado; Mrs. Ada Gregory Silor, Kansas, Mary Yeaton, Massachusetts.

RESEARCH: Hartsell Payne, Chairman, 510 Walnut St., Apt. 2, Terre Haute, Indiana; Dorothy Hitchcock, Michigan; Hazel Suessenguth, Ohio; Forrest Cross, Maryland; Elvyn Scott, Delaware; Ruth Guy, Texas.

MEMBERSHIP: Mrs. Lucille Wallace, Chairman, 1035½ Seventh Ave., North, Virginia, Minnesota; Lois Hensley, Florida; Sister Charles Miriam (Strassel), New Mexico; Winogene McIntyre, Washington; Edna Luneke, Michigan; Ina Maxson, Vermont.

FINANCE: Mary Eichian, 440 Lyceum Ave., Philadelphia 28, Pennsylvania; Louise Vance, Illinois; Oscar Stewart, Oklahoma; John Hannon, Ohio; Bernice Elliott, Nebraska; Loretta Laughlin, Minnesota.

LEGISLATION: Vernal Johnson, Chairman, 1115 Medical Arts Bldg., Oklahoma City, Oklahoma; Mrs. Evelyn Jardine, New Hampshire; Wilbert Zimmer, Ohio; Grace Mary Ederer, Minnesota; Beatrice Allison, New York; Barbara Isbell, California.

EDUCATION: Sister M. Antonia (Klaphecke), Chairman, Georgetown Hospital, Washington, D. C.; Joy Holm, Louisiana; Estelle Downer, Wisconsin; Mary Frances James, Alabama; Ruth Feucht, Illinois; Rose Hackman, Colorado.

STANDARDS AND STUDIES: Mollie Hill, Georgetown Hospital, Washington, D. C., Margaret Brown, Colorado; Sister M. Dolorosa (Pope), Missouri; Agnes Marie Lyman, Oregon.

CONVENTION COMMITTEE CHAIRMEN

GENERAL ARRANGEMENTS: Vondell Stewart, 3905 Day, Houston 6, Texas.

PROGRAM: Lucile Harris, Hendrick Memorial Hospital, Abilene, Texas.

EXHIBITS: TECHNICAL: Cecelia Korteum, 1164 North Dearborn, Chicago, Illinois. SCIENTIFIC:

REGISTRATION: Estelle Boyd, 406 Lott St., Yoakum, Texas.

SISTERS' RESERVATIONS: Sister M. Aniceta (Kelliher), St. Joseph's Infirmary, Houston 3, Texas.

PUBLICITY: Barbara Cleere, 2805 Rosedale, Houston 4, Texas.

ENTERTAINMENT: Doris Wallace, 8208 Jennings, Houston, Texas.

SPEAKERS' SUPPLIES: Sally O'Neil, 1723 Albans Road, Houston, Texas.

TRANSPORTATION: Ruth Diffey, 4717 Pershing, Houston, Texas.

PROGRAM COMMITTEE

We are striving to arrange an interesting scientific and educational 1950 Convention program which will meet the desires and needs of the majority of A.S.M.T. members and are soliciting your aid. There are, among our members, leaders in all phases of Medical Technology; therefore we would like to have a large contribution for our 1950 Program to come directly from our own membership. Those of you who are in a position to share your knowledge and experience with your fellow technologists, please do so by contributing a paper for our 1950 Convention Program. Read "Why Write a Scientific Paper," by Esther I. Wilbrecht in July AMERICAN JOURNAL OF MEDICAL TECHNOLOGY.

We have the State Paper-Writing Contest, the Convention Awards and the $200.00 Hilkowitz Memorial Contest to encourage each and every Medical Technologist to write a paper and we are looking forward and hoping to have a great many contributions.

Early notice of YOUR contribution for this occasion will be greatly appreciated. Also notify us of any suggestions or recommendations you want considered.

Please write Lucile Harris, Chairman, Hendrick Memorial Hospital, Abilene, Texas..

SCIENTIFIC EXHIBITS

In addition to the Scientific Exhibits as listed in the July JOURNAL, the following were presented at the convention in Roanoke, Virginia: these were con-competitive:

"American Society of Medical Technologists."

"Registry of Medical Technologists (ASCP)."

"Exfoliative Cytology of the Diagnosis of Malignant Neoplasma," C. Street, John T. Rogers, M.D., and George N. Papanicolaou, M.D., Cornell University.

"Parasitic Forms Encountered in the Clinical Laboratory," Edwin S. Gault, M.D., Temple University.

"Recommended Hematological Nomenclature," American Society of Clinical Pathologists.

"Vocational Guidance—Recruitment of Medical Technologists."

"Neuropathological Techniques Applicable to the Small Hospital Laboratory," C. Heinz and John A. Wagner, M.D., University of Maryland.

"The Baltimore Rh Typing Laboratory—A Community Health Service," E. Jahn and Milton S. Sacks, M.D., Baltimore.

With the excellence of the exhibits for the convention just past, we have proof of what is available, if we just think and plan ahead a bit. Why not prepare YOUR exhibit for the 1950 meeting? Begin to think about it now. Yours may be just the type of work which will lend itself to be shown other members of the national society. Why not plan now what you would like to present?

MISCELLANEOUS ANNOUNCEMENTS

The KANSAS Society of Medical Technologists held a meeting on July 10, at the Veterans' Hospital, Wichita. Miss Dolores Mans, Delegate to the A.S.M.T. Convention in Roanoke, Va., reported on the meeting and exhibited material presented during the convention. The movie, "Bone Marrow," from Armour & Co., was shown. Kansas University will hold a three-day Seminar, in Lawrence, on January 23, through 25, 1950.

THE EXECUTIVE OFFICE
of the American Society of Medical Technologists
and the
EDITORIAL AND BUSINESS OFFICE
of the American Journal of Medical Technology

are now located at 6544 FANNIN ST., HOUSTON 5, TEXAS. Please check your name and address as they appear on the journal envelope, and send any corrections or changes to the above address promptly. If your journals are delayed, it may be because you have failed to let us know your new address. Just place the following information on a penny postcard and mail to us: print (or type) your name—if this is changed, give your maiden name, as we have it in our files, in parentheses, as: Mrs. Jane (Doe) Roe, *old address*, changed to *new address*. Be SURE TO GIVE BOTH THE OLD and NEW addresses, and designate which is which.

DUES are payable NOW. Send your annual dues promptly to your local (district) or to your state treasurer.

The MEMBERSHIP COMMITTEE is already hard at work to add 1500 new members to A.S.M.T. before January 1, 1950. How about doing YOUR PART, and help them get just one more? Tell just one registered medical technologist whom you know is not already a member of his professional organizations, how it is to his advantage to BELONG.

The A.S.M.T. exhibit is available to state societies for their conventions. Why not book it NOW for your State Hospital Association meeting, also. Help your newly organized Public Relations Committee in this way, to "let the people know" what Medical Technology is—and what the American Society of Medical Technologists stands for.

Your Executive Office stands ready to serve you, as medical technologists, and members of your professional organizations. Your problems will be forwarded to the committee best able to deal with them. If these problems relate to matters concerning training schools, they will be forwarded to the A.M.A. Council on Education; if to qualifications and certification of medical technologists, the Registry will be the recipient. Your Executive Office will be the clearing house, and will function conscientiously to give your concerns prompt attention. Be sure to note the address: 6544 Fannin St., Houston 5, Texas. Any member of A.S.M.T., any registered Medical Technologist, any "would-be" Medical Technologist, any "friend" of Medical Technology, will ever be welcome at that address. We are looking forward to having most of you "drop in" when you come to the convention next year, so begin making your plans now.

LOAN SETS

Hematology Loan Sets available. The deposit has been reduced to $10.00, $8.50 of which will be returned when the set is received intact from the borrower. Loan sets may be kept for two weeks. Write the Executive Office, American Society of Medical Technologists, 6544 Fannin St., Houston 4, Texas.

REFRESHER COURSES

Laboratory Diagnosis of Parasitic Diseases (6 wks.)
From September 12 to October 21, 1949.
Laboratory Diagnosis of Bacterial Diseases, Part 2, General Bacteriology (5 wks.)
From October 31 to December 2, 1949.
Laboratory Diagnosis of Rabies (1 wk.)
From November 14 to November 18, 1949.
Advanced Enteric Bacteriology (2 wks.)
From December 5 to December 16, 1949.

Information and applications should be requested from the Chief, Laboratory Division, Communicable Disease Center, 291 Peachtree Street, N. E., Atlanta, Georgia.

R. F. Reider, Senior Surgeon in Charge, Special Services.

The graduate program in Medical Technology at Georgetown University Medical Center, Washington, D. C., is now fully organized and will begin this fall.

In November, 1949, it is the plan of the Washington University School of Medicine, St. Louis, Missouri, to offer a postgraduate course to medical technologists. This year it will probably extend over a period of three days. Details are not completed at the present time, but anyone interested may make direct communication with Dr. Gustave J. Dammin, M.D., Washington University, Department of Pathology, Euclid Ave. and Kingshighway, St. Louis 10, Missouri. (See notice on advertising pages this issue.)

NOTICE TO ALL MEDICAL TECHNOLOGISTS

The Board of Registry will meet October 8 and 9 in Chicago, Drake Hotel. The Board wishes to inform the medical technologists that this meeting is entirely for the benefit of the Registered Medical Technologists and the various problems they may have. In order that the pathologists of the Board know our ideas and our wishes the Board allows two members to be "sit-in members" and to discuss the various questions and problems that have arisen and will possibly arise. The second day of the meeting three technologists representing the Registered Tech-

nologists at large meet also with the Board and hear what has taken place and offer suggestions.

In order that the technologists be good representatives they must hear from the technologists throughout the entire country, as many problems are local in nature and have not been problems in other areas. In making the agenda for the meeting and making the questions that need to be asked we strongly urge you to ask specific questions and also give us any information that is of importance or will become a problem. We urge you to write this to the technologists on the Board. This is your chance to act to clarify any point and if it so happens you have tried a specific idea in your school or organization of technologists, the Board of Registry will welcome that idea.

Write the question or the point of information to any one of the Medical Technologists listed below. Be sure in all instances to make your points clear.

Rachel Lehman, M.T. (ASCP), (Chairman)
3939 North Capitol Avenue
Indianapolis, Indiana

Ida Reilly, M.T. (ASCP)
Roanoke Hospital Association
Roanoke, Virginia

Lucille Wallace, M.T. (ASCP)
1035½ Seventh Avenue, North
Virginia, Minnesota

STATE MEETINGS

Wisconsin Association of Medical Technologists—St. Francis Hospital, LaCrosse, October 22, 23, 1949.

Washington State Society of Medical Technologists—October, 1949.